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THE EYE
AND
NERVOUS SYSTEM

THEIR DIAGNOSTIC RELATIONS
BY VARIOUS AUTHORS

EDITED BY

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PREFACE

ALTHOUGH it is generally conceded that a knowledge of neurology is indispensable to the ophthalmologist and that an acquaintanceship with ophthalmology is of the greatest service to the neurologist, there is no book in the English language which covers the ground where these two specialties meet. Several German works and one French book have appeared which include much that may be found in these pages, but even the student who is conversant with these languages will frequently find these books lacking in the information he seeks, and he will often be compelled to search through many other sources of information before he obtains what he desires. It has been the aim of the editors, therefore, to present under a single cover the phases of ophthalmology and neurology which are in any way connected with each other, hoping thereby not only to facilitate the labors of those practising these specialties, but also to present ophthalmo-neurology in such a form that the general practitioner may without difficulty become conversant with its symptomatology.

While it may be impossible for a physician in general practice to be at the same time an ophthalmologist and a neurologist, it is quite possible, and indeed essential, that he should be aware of the extent, as well as of the limitation, of the knowledge of these two specialties and of the aid they may offer him in the recognition of disease. So many general morbid processes are announced by some minute change in the nervous system, and such unusual opportunities of studying alterations in the vascular system are presented in the eye, that the value of a clear understanding of disease of these structures cannot be overestimated, and the necessity for proper training in the methods which lead to its recognition, cannot be too strongly insisted upon.

Realizing the extent of the task before them, and the increased value which the work would have if it represented the views of others as well as their own, the editors have been most fortunate in securing a number of distinguished American ophthalmologists and neurologists as collaborators, so that the volume consists of a series of monographs by authors peculiarly well fitted by previous study and investigation to treat authoritatively of the subjects assigned to them.

In dividing the subjects into the various chapters, care was taken to avoid repetition of the same matter by different authors. In the chapters where overlapping does occur, it was done purposely, with the idea of having both the ophthalmological and the neurological phases fully presented.

The editors desire to express their thanks and deep sense of appreciation to their contributors for the very admirable way in which they have treated the subjects allotted to them. It has been a source of great regret that the work did not appear, as originally planned, some six months ago, but, despite every effort of the publishers, certain labor difficulties prevented, and it has been impossible to issue it until now.

The index has been prepared by Dr. Paul La Roque and the editors would express to him their thanks for his thoroughness.

THE EDITORS.

PHILADELPHIA, April 10, 1906.

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THE
EYE AND NERVOUS SYSTEM

CHAPTER I.

THE INTRACRANIAL PORTION OF THE ENCEPHALIC NERVES, ESPECIALLY OF THOSE RELATED TO THE EYE.

BY WILLIAM G. SPILLER, M.D.

THE OLFACTORY NERVE.

THE olfactory, or first, nerve has its origin in the regio olfactoria of the Schneiderian membrane. After the fibres constituting this nerve have entered the cranium through the cribriform plate, they penetrate the olfactory bulb and terminate in the olfactory glomeruli. The olfactory nerve, therefore, has a very short intracranial course, its intracranial connections belonging to the very complex central system of fibres of the olfactory apparatus.

THE OPTIC NERVE.

The optic, or second, nerve arises and terminates within the retina, and the structure commonly called the optic nerve is in reality a part of the brain.

Decussation of the Visual Fibres in the Optic Chiasm.—Were it not for the opinion of Kölliker, probably there would not have been so much doubt concerning the decussation in man of the nerve-fibres in the optic chiasm. Kölliker believes that the decussation is complete, and his views have justly received much critical attention. The subject has been presented clearly by Warrington and Dutton in a contribution by which they try to show that Kölliker is in error, and in this attempt they are aided by the investigations of many others. Kölliker admits the existence of a small number of direct fibres which pass to the tract of the same side as the optic nerve from which they are derived. He is not alone in his views because, according to Warrington and Dutton, Biesiadecki in 1861, Mandelstamm in 1873, Scheel, W. Krause in 1897, and Michel, 1873 to 1890, also have favored the view that the decussation is complete. Michel's argument that if homonymous bilateral hemianopsia is to be explained by the decussation in the chiasm, the proportion of crossed to uncrossed fibres should be as 1 : 1, and not, as it is admitted to be by the adherents of a partial crossing, as 3 : 1, or 4 : 1, or even as 5 : 3, is answered by Warrington and Dutton. In a case studied by them there was not any marked difference between the amount of degeneration in the two optic tracts, although only one optic nerve was diseased. Furthermore, they point out, that owing to the limitation of the field of vision, the outer and lower parts of the retina are probably less developed than the inner and lower, and, as Michel himself has found, the number of fibres passing to different sections of the retina is very variable, being least in the outer segment. They refer to Michel's statement that no anatomical evidence has yet been produced demonstrating the double representation of the macula in the cerebral cortex, which is required by the theory of semi-decussation in order to explain the freedom of the fixation point in cases of hemianopsia, and they remark that Michel's hypothesis cannot be regarded as giving a more satisfactory explanation. They refer also to Grützner's work on this subject. This investigator formed models of the chiasm in which one-half of the fibres were made to cross and the other half to remain on the same side. The models were then embedded in paraffin and cut into a series of horizontal sections. From a study of these sections Grützner concluded that if he had not known that the decussation was partial he would have supposed it to be complete.

Warrington and Dutton's case was one in which enucleation of the right eyeball had been performed on account of trauma. The chiasm with the optic nerves attached was cut in a complete series of about 500 horizontal sections. In the sections about the median plane the atrophy from the right nerve was seen spreading in the central portion of the same-sided tract, and occupying the front and back of the chiasm, its extent being larger in the latter situation. Even though Michel has shown that the fibres of the optic nerve, as they enter the chiasm, turn sharply at a right angle, and again at another right angle when entering the tract of the opposite side, Warrington and Dutton believe they were able to detect groups of fibres passing along the outer border of the nerve and into the tract of the same side; and they state that all observers (Purtscher, Wieting, Siemerling, Williamson) with the modern methods, with the single exception of Michel, are of the opinion that degeneration occurs in both optic tracts after destruction of one optic nerve; and the crossed fibres usually occupy the centre of the tract, the uncrossed the periphery.[1]

The findings that Probst obtained in his experiments on the optic fibres resemble very closely those of von Monakow. Probst believes that the decussation of the optic fibres in the dog and cat is a partial one. The medial-central fibres of the optic nerve cross first, and later the dorsal and ventral fibres, while the lateral fibres do not decussate. Most of the optic fibres cross to the opposite optic tract.

When an eyeball is removed degenerated optic fibres are found in both external geniculate bodies, but more are found in the opposite ganglion.

The arm of the anterior colliculus of the corpora quadrigemina contains chiefly crossed, but also uncrossed, optic fibres.

In studying the optic chiasm of a new-born child, stained by the hæmatoxylin method of Weigert, a partial decussation seems to me very probable. The course of the nerve-fibres in the optic nerves, chiasm, and optic tracts may be better seen in specimens obtained from a new-born child than in those from an adult, because the number of stained fibres is less in the former. A careful study of the literature on decussation of the optic fibres seems to show that the weight of evidence is in favor of a partial decussation, and in support of this view are the important studies of Ramón y Cajal.

Parsons, from his investigations on monkeys, concludes that, speaking generally, the fibres derived from various parts of the retina retain their relative position in the optic nerve,—*i.e.*, temporal fibres remain external, nasal internal. The temporal fibres tend towards the dorsal side as they approach the chiasm, and the nasal towards the ventral side. A further dislocation, affecting the temporal fibres most, occurs as the result of the intercalation of the papillomacular bundle. There may be some variation in different individuals.[2]

According to Henschen, as given by Wilbrand and Saenger, the uncrossed bundle (not papillomacular fibres) near the eyeball is divided into two parts, between which are placed the fibres of the papillomacular bundle, more centrally the two portions of the uncrossed bundle approach one another and in the posterior half of the optic nerve in transverse section unite and assume a sickle shape and are situated in the ventrolateral portion of the nerve. The crossed bundle (not papillomacular bundle) has a dorso-medial position throughout the optic nerve.

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[1] Warrington and Dutton, Brain, 1900, vol. xxiii., No. 92, p. 642.

[2] Lancet, April 16, 1904, p. 1029.

The structure of the chiasm is very complicated. Probably the most important fact is that the uncrossed fibres are mingled with the crossed in the lateral portion of the chiasm, so that nasal hemianopsia can not be caused by a lesion of the chiasm.

There is much difference of opinion, as shown by Wilbrand and Saenger, regarding the existence of a separate bundle of uncrossed fibres in the optic tract, some believing that the crossed are mingled with the uncrossed fibres. Henschen is of the opinion that the uncrossed bundle occupies the superior and external position and is distinct from the crossed fibres.

Decussation of the Macular Fibres.—Dean and Usher have attempted to determine the position of the optic nerve-fibres in monkeys. Vossius, in a case of symmetrical central scotoma in man, found that the macular fibres lie close under the dorsal margin of the chiasm. One of the cases reported by Dean and Usher corresponds closely with Vossius's description. Bunge found, they say, that the papillomacular bundle maintains a dorsal position throughout the chiasm in man. Dean and Usher give the course of the macular fibres in the chiasm in the monkey as follows: Anteriorly in the chiasm they occupy the central area of both halves. Soon after entering the chiasm they pass towards the dorsum before any crossing has occurred. The crossing takes place first at the dorsal periphery, at some distance behind the front of the chiasm. On proceeding backward, though still partly remaining dorsal, they spread from the dorsal surface in a ventral direction. The macular fibres never reach the ventral periphery. These investigators were unable to determine whether the macular fibres cross wholly or partially in the chiasm, as they could not be certain whether the wound was limited to the macular fibres, or whether it had damaged only a portion of these.[3]

According to Henschen, as quoted by von Monakow, the bundle from the macula lutea between the retina and the primary optic centres lies in the middle of the transverse sections of the optic nerve and optic tract, whereas the rest of the retina is represented in the periphery of these structures.

Wilbrand and Saenger describe the course of the papillomacular bundle according to Uhthoff as follows: In the forward portion of the optic nerve the bundle lies in wedge form in the outer and lower part

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[3] Dean and Usher, Brain, 1903, vol. xxvi., No. 104, p. 524.

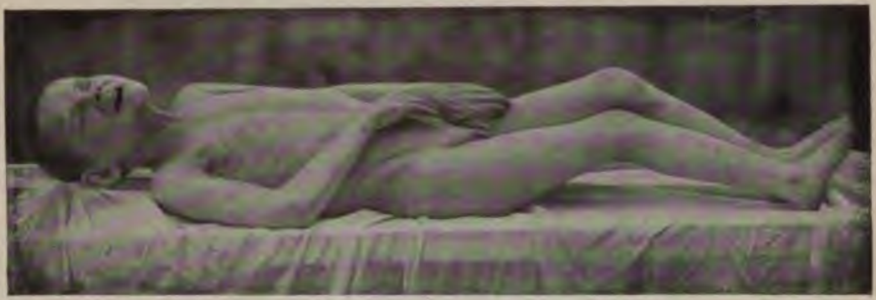

FIG. 1.—Case of complete absence of the visual system: other parts of the body, except the external genitalia, were well developed. The age of this patient was twenty-two years. The limbs were spastic.

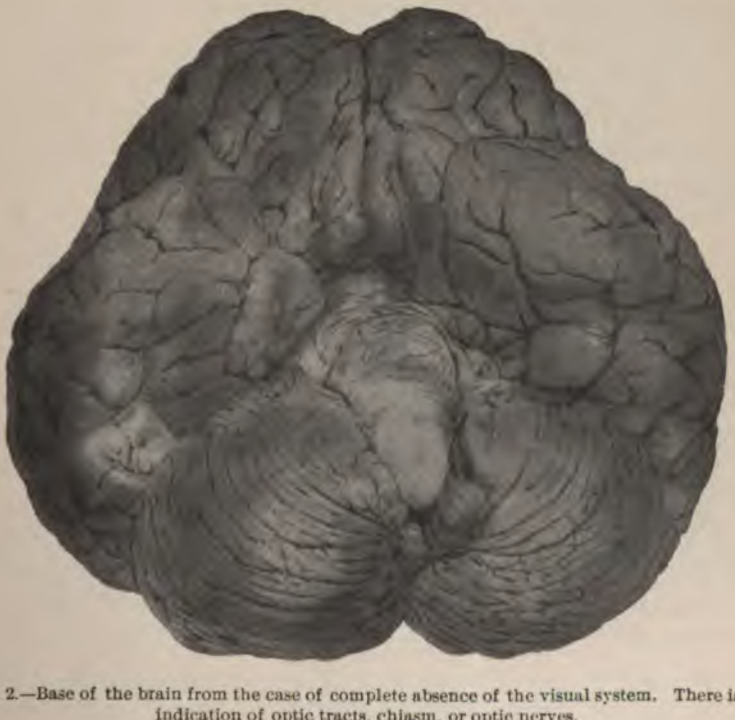

FIG. 2.—Base of the brain from the case of complete absence of the visual system. There is no indication of optic tracts, chiasm, or optic nerves.

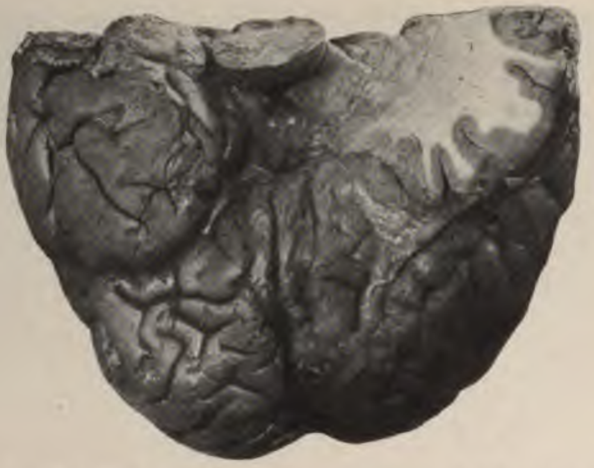

FIG. 3.—Portion of the brain from the case of complete absence of the visual system. A part of the right temporal lobe has been cut away, in order to show the absence of the optic tract and external geniculate body. The internal geniculate body is well developed.

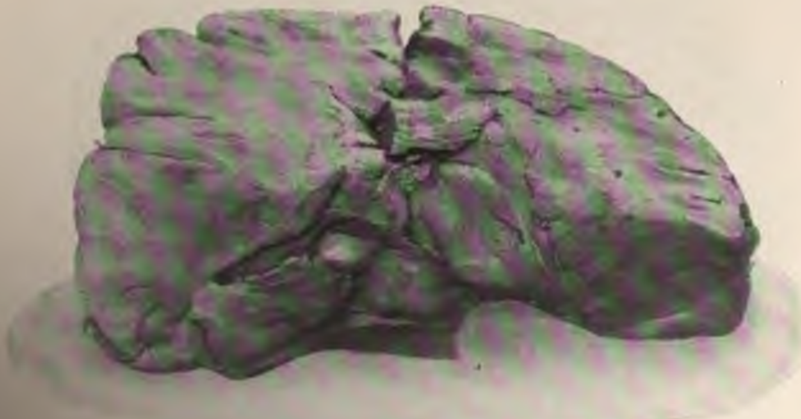

FIG. 4.—Portion of the brain from a case of complete absence of the visual system. The pulvinar is of good size on each side. The anterior colliculi of the corpora quadrigemina appear smaller than the posterior, because they were further from the camera.

of the nerve, with the point of the wedge toward the central vessels, and from here posteriorly gradually assumes a crescent shape, the ends of the crescent withdrawing from the periphery. After the exit of the central vessels from the optic nerve the bundle gradually assumes a vertically oval form, and becomes centrally placed in the posterior orbital part of the nerve. There are variations in different cases as regards the point at which the bundle becomes central. The bundle is central also in the intracranial part of the optic nerve, but because in this portion the nerve is more oval the bundle has a greater transverse than a vertical diameter. In the tract the bundle occupies the centre.

PRIMARY OPTIC CENTRES.

The fibres of the optic nerve end chiefly in the external geniculate body, but some pass to the posterior part of the optic thalamus known as the pulvinar and to the anterior colliculus of the corpora quadrigemina. The fibres to the latter structure in man cannot be numerous, although they are in the rabbit. In a case studied by me in which the visual system was absent in an adult there was no external geniculate body and no trace of either optic nerve; the anterior colliculi of the corpora quadrigemina and the pulvinar on each side were well formed.

The external geniculate body, the anterior colliculus of the corpora quadrigemina, and the pulvinar are known as the primary optic centres, and from these bodies other fibres pass to the visual cortex of the brain, although they contain also the ramifications of fibres that arise in the visual cortex (Edinger). The optic tract is formed chiefly by nerve-fibres that terminate in the primary visual centres, but according to Edinger they contain also, certainly in the rabbit and cat and probably in man, fibres that arise in the corpora quadrigemina. These fibres from the primary visual centres terminate in the retina. According to von Monakow as much as 80 per cent. of the visual fibres in man terminate in the external geniculate body, and all the visual fibres of the optic tract terminate in the primary visual centres. Von Monakow is authority for the statement that hemianopsia as the result of a lesion confined to the anterior colliculus of the corpora quadrigemina has not been observed, and it is doubtful whether a lesion confined to the pulvinar and not implicating the external geniculate body causes hemianopsia.

Some optic fibres pass above and below the external geniculate body as stratum zonale to the pulvinar of the optic thalamus and near to the ganglion habenulæ, with which, however, they do not come in contact (Probst).[4]

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[4] Probst, Monatsschrift für Psychiatrie und Neurologie, September, 1900, vol. viii. p. 165.

"PUPILLARY FIBRES" IN THE VISUAL SYSTEM.

Von Gudden showed that the optic nerve contains thick and slender nerve-fibres, and, according to von Monakow, the former pass almost entirely to the anterior colliculi of the corpora quadrigemina. These thicker fibres are supposed to serve a special function,—viz., the contraction of the iris in the light reflex. They have been recently studied by Reichardt. It is remarkable that these "pupillary fibres" should be present in the optic nerve and optic tract, and although they have not been actually demonstrated much evidence of their existence has been collected. They were first recognized by von Gudden and von Bechterew, and are believed to have no part in vision. Their function is to convey centripetal impulses to the pupillary reflex centre wherever that is located. The results obtained by Massaut in his study of the "pupillary fibres" are so extraordinary that they may be referred to in this connection. This investigator extirpated the iris in rabbits, and found degenerated fibres by the Marchi method in the optic nerve, which he regarded as "pupillary fibres." The interpretation of this degeneration offered by him is accepted with some difficulty, inasmuch as a degeneration of one group of fibres, the centripetal, is supposed to have occurred as a result of a lesion of the distal ends of another group of fibres, the centrifugal. This, in my experience, is so unlike what occurs elsewhere in the nervous system that I can accept Massaut's interpretation only with much hesitation, especially as it is well known that the Marchi method is at times the source of many erroneous conclusions. (See chapter vi., p. 291.)

Westphal found that the large fibres in the optic nerve receive their medullary sheaths sooner than the fine fibres, and he also concluded that the former are "pupillary fibres." Reichardt points out that disease of the optic nerve may cause complete blindness and yet leave the iritic reflex intact, and this seems to indicate that there are distinct systems of fibres in the optic nerve. In compression of the optic nerve the iritic reflex is much less affected than vision, which may indicate that the "pupillary fibres" are more resistant. When acute inflammation of the optic nerve is disappearing improvement is usually noticed in the reaction of the iris before vision begins to return.

Piltz describes a case in which blindness had existed nine years and was caused by optic atrophy, and yet the iritic response to light was preserved. Bach observed a case of tabes in which the reflex action of the iris to light was better in the eye in which vision was more affected and the optic nerve atrophy was more advanced. All this seems to indicate that there are probably special fibres in the optic nerv the iritic reflex. Reichardt also has observed an important case. A man had atrophy of the left optic nerve, and until four weeks before the loss of sight in the left eye became complete he had normal pupillary reaction, and even later the reaction was only somewhat tardy. The left optic nerve was found to be small, and the reduction in size was caused chiefly by the degeneration of the fine fibres. This case seems to afford some support for the widely accepted teaching that the fine fibres especially are concerned with vision.

Reichardt believes that the " pupillary fibres" of the optic nerve may alone degenerate, or at least be more affected, and in such a case vision would be preserved although the iritic reflex would be lost.

According to Bach and Meyer, it is probable that the pupillary reflex may be stimulated from any part of the retina, although the macula region is probably the most important. The " pupillary fibres" are mingled with the visual fibres in the optic nerve, although Bach believes a certain order is preserved, so that those fibres that come from the temporal side of the retina remain in the temporal side of the optic nerve. In man the decussation of the " pupillary fibres," as is that of the visual fibres, is partial, although most of the fibres of each variety cross. The visual and " pupillary" fibres are associated in the optic tract as far as the external geniculate body, but here they separate.

It may be said that no one has ever been able to trace the connection between the optic nerve and the oculomotor nucleus. It has been supposed that this connection is made by means of Meynert's fibres passing in a curve below the aqueduct of Sylvius. Some authors have accepted this view as an established fact, forgetting or being in ignorance of the uncertainty concerning this subject. No one disputes that these fibres of Meynert from their position might serve as the connecting link, but there are other fibres in this region which might have this function.

Probst, whose anatomical studies of the nervous system are so well known, says that when an eyeball has been enucleated degenerated fibres of the optic nerve cannot be followed to the oculomotor nucleus. The connection of the optic fibres with the oculomotor nucleus must therefore be by means of a short system of fibres (Schaltsystem).

It is supposed that the " pupillary fibres" pass from the optic tract to the corpora quadrigemina through the arm of the anterior colliculus of this body, but the further course of these fibres is unknown. Bernheimer believes he has been able to trace these fibres to the region below the aqueduct of Sylvius and near to the oculomotor nucleus, but Dimmer, Bach, and others have followed the fibres only so far as the arm and the upper part of the anterior colliculus. The "pupillary fibres" are believed by some to be in relation with the stratum zonale of the pulvinar of the optic thalamus, with the corpus hypothalamicum, and with the ganglion habenulæ, but this relation seems improbable.

Edinger, in the latest edition of his lectures upon the anatomy of the nervous system, expresses himself as in doubt concerning the connection of the "pupillary fibres" of the optic nerve with the oculomotor nerve. While acknowledging that the "pupillary fibres" probably pass through the anterior arm of the corpora quadrigemina, he is uncertain concerning the manner of their connection with the oculomotor nerve. Von Monakow also is cautious concerning this supposed connection.

THE CORTICAL CENTRES OF VISION.

One of the earliest cases, if not the earliest case, of bilateral hemianopsia from a unilateral lesion was that published by Lewick;[5] the lesion was an abscess of the right occipital lobe.

Henschen believes that only a lesion of the cortex of the calcarine fissure, and especially of its anterior two-thirds, causes persistent bilateral homonymous hemianopsia. Lesions of other parts of the cortex of the occipital lobe may exist without such visual disturbance. Von Monakow says that Huguenin assumed that destruction of the cortex of the calcarine fissure causes hemianopsia. Later investigators extended the visual cortex; von Monakow at first included the cuneus, lingual lobule, and descending gyrus. Seguin limited the visual cortex to the cuneus; Nothnagel limited it to the cuneus and first occipital gyrus; Vialet to the entire medial part of the occipital lobe; Ferrier, Angelucci, Bianchi, Reinhard, Luciani, Seppilli included the cuneus and the medial part of the occipital lobe; the first extended it to the third occipital gyrus and even the angular gyrus.

Henschen says that all the lateral surface of the occipital lobe and the angular gyrus may be destroyed without causing hemianopsia. In one of his cases a lesion was confined to the calcarine cortex, and yet hemianopsia was caused by this lesion. In another case the calcarine cortex was destroyed and hemianopsia was caused thereby, while extensive destruction of the lateral surface of the other hemisphere, presumably the occipital lobe is meant, did not cause hemianopsia. The cases Henschen refers to seem to show that anopsia in the lower quadrant may be caused by a lesion of the dorsal part of the external geniculate body, and that in such cases normal fibres may be traced from the lower part of the external geniculate body to the inferior lip of the calcarine fissure; by means of these fibres are conveyed the impressions made on the inferior part of the retina. His observations seem to show that the fibres of the superior lip of the calcarine fissure are in connection with the superior part of the retina. Henschen combats Ferrier, who places the visual centre in the angular gyrus, and Nothnagel and Munk, who regard the first occipital convolution as part of the visual centre. He has observed anopsia of the superior quadrant caused by softening of the inferior lip of the calcarine fissure. In another case horizontal scotoma was caused by a lesion confined to the base of the calcarine fissure.

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[5] Amer. Jour. of the Med. Sci., 1866.

These investigations of Henschen are extremely important, and seem to show that the upper part of the retina is represented in the upper part of the external geniculate body and in the upper lip of the calcarine fissure; that the lower part of the retina is represented in the lower part of the external geniculate body and in the lower lip of the calcarine fissure; and that the horizontal zone of the retina, midway between the upper and lower portions, is represented in the base of the calcarine fissure. From this it follows that the fibres from the different portions of the retina in the optic nerve and optic tract must preserve their relative positions.[6]

The case of quadrant anopsia, recently reported by Beevor and Collier (Brain, 1904), with necropsy and careful microscopical study, is exceedingly valuable in determining the location of the cortical lesion causing quadrant anopsia. A case similar to this has been reported by Hun. The only symptom of nervous disease presented while Beevor and Collier's patient was under observation was blindness of the left upper quadrants of both visual fields, the fixation point escaping. This condition remained constant nearly two years. An occlusive lesion of the right posterior calcarine artery had caused destruction of the cortex (1) of the right fusiform lobe for its posterior two centimetres, (2) of the right lingual lobe from the junction of the calcarine and parieto-occipital fissures to the pole of the hemisphere, (3) of the whole cortex in the depth of the calcarine fissure, (4) of the greater part of the inferior cuneal gyrus, small areas only at the anterior and posterior limits of this gyrus being free. The necrosis did not involve the optic radiations at any point. The only parts of the cortex of the mesial aspect of the occipital lobe which had escaped destruction were the upper two-thirds of the cuneus and the anterior and ventral portion of the fusiform gyrus. The lower quadrants of the visual fields were entirely unaffected. The case seems to show that the cortex of the upper two-thirds of the cuneus is the visual centre for the lower quadrants.

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[6] Henschen, Semaine Médicale, April 22, 1903, No. 16, p. 125.

In this case the cortex lining the calcarine fissure was completely necrotic. The primary half-vision centre, the authors think, cannot therefore be limited to the calcarine cortex, nor the macula exclusively represented in the anterior part of this region. The sharp distinctions of the upper and lower fields as related to the calcarine fissure, made by Henschen would seem by this case to need some modification, for the lower part of the retina would appear to be represented in the upper lip of the calcarine fissure.

Harris[7] thinks it is possible to have a loss of the lower quadrant on the one side and of the upper quadrant on the other from a lesion in each cuneus, and he says such a case has been recorded by Weymann, but how the function of only one quadrant of the retina of one eye can be lost by a lesion of the visual cortex is to me unintelligible. Even granting that the crossed and uncrossed fibres remain separate so far as the optic tract, we have no proof that they are ununited in the optic radiations and visual cortex. A lesion in these parts seems to cause disturbance of vision in each eye.

Wilbrand thinks that every part of the macula is represented both in the right and left visual cortical areas, but in cases in which the dividing line in hemianopsia passes directly through the fixation point the double representation does not occur. It has also been suggested (Förster) that the macula region of the cortex is better supplied with blood-vessels than other parts of the visual cortex, and receives nourishment through the posterior cerebral artery and the occipital branch of the artery of the Sylvian fossa. Von Monakow thinks that the representation of the macula is not confined to the middle of the visual centre, nor to its periphery, but he believes that all parts of the occipital lobe and possibly even the posterior part of the angular gyrus are represented in the macular field, and that the macular field may extend far beyond the limit of the area usually recognized as the visual region of the cortex. He does not accept Henschen's views that the macula is represented in the anterior portion of the cortex of the calcarine fissure.

Von Monakow says that a careful examination of the reported cases shows that hemianopsia is most likely to occur and to be persisting perhaps only from a lesion in the calcarine fissure, but there are some cases in which hemianopsia has occurred from lesion elsewhere, although it may be possible that in these cases the hemianopsia was caused by implication of the optic radiations, but the same is true when the lesion is in the calcarine fissure. Von Monakow thinks that in every persisting cortical hemianopsia there is partial injury of some part of the occipital lobe and—which is more important—also a lesion of the optic radiations.[8]

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[7] Brain, vol. xx., 1897, p. 308.

Few anatomists place the visual centre in the angular gyrus, but there is more doubt as to the implication of the lateral surface of the occipital lobe in the visual field, and as to the extent of the surface involved.

Gallemaerts has found that after enucleation or atrophy of an eyeball the cells of the visual cortex become less numerous, and this is to be seen in both occipital lobes after a lesion of one eyeball. This finding confirms the partial decussation at the optic chiasm. The crossed fasciculus contains more fibres than the direct fasciculus; therefore the greatest diminution of nerve cells is in the visual cortex opposite to the side on which the eyeball has been lost.[9]

From a study of the diminution in the number of nerve-cells Gallemaerts concludes that the fusiform lobule, the angular gyrus, and the descending gyrus (which is on the outer surface of the occipital lobe and at the occipital point) are not part of the visual centre, but that the visual centre consists of the lingual lobule and the cuneus. In these convolutions, especially near the calcarine fissure, the lesions were very distinct. The diminution of the nerve-cells in the visual cortex after destruction of the eyeball occurs only when years have passed since the lesion developed, and all the layers of nerve-cells in the visual cortex are affected after a sufficiently long period.

Bernheimer includes the cuneus, the cortex of the calcarine fissure, the lingual lobe, and the descending gyrus in the visual cortex.

The reader is referred to the article by Dr. Charles K. Mills for further remarks on the lower and higher cortical visual areas.

THE OCULOMOTOR NERVE.

The nucleus of the oculomotor, or third, nerve lies below the aqueduct of Sylvius. This group of cells consists of a median nucleus with a lateral nucleus on each side of it. Anterior to these a group of small cells is found on each side of the median line, and is known as the

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[8] Von Monakow, Gehirnpathologie.

[9] Gallemaerts, Bulletin de l'Académie Royale de Médecine de Belgique, 1902, iv. series, vol. xvi. p. 267.

Edinger-Westphal nucleus. It has been considered as the source of the innervation of the inner muscles of the eyeballs, but there is doubt whether it should be regarded as a part of the oculomotor nucleus. The fibres of the oculomotor nerve pass ventrally through the tegmentum of the cerebral peduncle and reach the surface at the inner border of the foot of the cerebral peduncle (crusta). The individual fibres forming this nerve are not closely united even at the exit of the nerve from the cerebral peduncle, and it is because of this that paralysis of one or more of the muscles innervated by the oculomotor nerve may be caused by basal meningitis without paralysis of the whole muscular supply of this nerve, and such paralysis may mistakenly be attributed to nuclear disease.

There is some doubt whether the muscles of the eye innervated by the oculomotor nerve are separately represented in the nucleus of this nerve. The knowledge concerning this subject that has been obtained depends chiefly on investigations upon monkeys. Siemerling believes that the investigations of Bach, Bernheimer, van Gehuchten, and van Biervliet have not led to uniform results. The oculomotor nucleus, according to Siemerling,[10] is merely the place for the transference of impulses, and we are not in a position to state which parts are concerned with the innervation of the individual muscles. Edinger's views as contained in the seventh edition of his lectures upon the structure of the nervous system are as follows: The most frontal group supplies the levator palpebræ and rectus superior of the same side. From the caudal portion arise the fibres for the rectus inferior and the trochlearis of the opposite side. The intervening cells give origin to both direct and crossed axis cylinder processes, so that each obliquus inferior and each rectus internus receives fibres from the right and left lateral group of cells. The nerve-cells for the rectus internus are on each side so near the large cell median nucleus that Edinger believes the three groups together may form a centre for convergence. All the external ocular muscles he believes are supplied from the nuclei of the oculomotorius, trochlearis and abducens. The fibres for the inner ocular muscles (iris and ciliary muscle) arise in the ciliary ganglion, and fine oculomotor fibres terminate in this ganglion; possibly these fibres arise in the small cell frontal groups.

Edinger says it is not certain whether fibres pass directly from the oculomotor nucleus to the iris, or whether they are interrupted in the ciliary ganglion; the latter seems the more probable. Bernheimer has caused paralysis of the iris in the monkey by a lesion of the frontal portion of the oculomotor nucleus.

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[10] Archiv für Psychiatrie, vol. xl., No. 4, p. 61.

The connection of the cortical centres with the nuclei of the ocular nerves has not yet been determined, and the location of the cortical centres is still very uncertain.

Marina's investigations have made it doubtful whether a centre for convergence in the oculomotor nucleus exists. He has shown that when another ocular muscle was transplanted and allowed to unite with the tendon of the internal rectus, contraction of the pupil in convergence still occurred, although the internal rectus muscle had no part in the convergence movement of the eyeball. Contraction of the pupil occurred when the eyeball was drawn inward through electrical irritation of the internal rectus, and even when it was drawn inward by forceps without muscular action. All these experiments seem to show that there can be no centre for convergence.[11]

Van Gehuchten and van Biervliet, from their studies on rabbits, conclude that the uncrossed fibres of the oculomotor nerve arise chiefly in the ventral portion of the nucleus and the crossed fibres in the dorsal part of the nucleus. The upper end of the nucleus, therefore, gives origin only to direct fibres. The nerve-cells scattered between the fibres of the posterior longitudinal bundle (the lateral cells of Bernheimer, the aberrant cells of Biervliet) belong to the oculomotor nucleus and give origin to direct fibres.

Bernheimer[12] believes that the muscles of the eyeballs are innervated from the lateral nucleus (chief nucleus) and that the median nucleus and the Edinger-Westphal nucleus innervate only the inner muscles of the eye.

The middle portion of the lateral nucleus, which gives origin to crossed fibres, according to this investigator, innervates the inferior rectus: further forward is the nucleus of the internal rectus of the same side. The lateral portion of the nucleus gives origin to fibres for the internal rectus. The group of cells innervating the superior rectus is further forward than that innervating the internal rectus. The frontal portion of the lateral nucleus contains the nucleus of the levator palpebræ superioris. Distal to the lateral nucleus is the group of cells innervating the inferior rectus and bordering on the nucleus of the trochlear nerve. It gives origin only to crossed fibres.

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[11] Marina, Neurologisches Centralblatt, September 1, 1904, p. 797.

[12] Bernheimer, Neurologisches Centralblatt, 15, 11, 1899.

$$\textbf{Chief Nucleus}\left\{\begin{array}{l}\textbf{Levator palpebræ}\\ \textbf{Rectus superior}\\ \textbf{Rectus internus}\\ \textbf{Rectus inferior}\\ \textbf{Obliquus superior}\end{array}\right\}\textbf{Median Line}$$

Most writers agree that the Darkschewitch nucleus is not a part of the oculomotor nucleus. No scheme of the divisions of the oculomotor nucleus can as yet be accepted as final.

I cannot believe that the centre for the superior rectus is so far forward as is usually represented. In two cases of tumor under my observation the upward movement of the eyeballs was much more affected than the downward movement. A necropsy in each case revealed a tumor of the pons.

Some investigators doubt the existence of a pupillary reflex centre in the oculomotor nucleus, and they point out that no explanation for reflex pupillary rigidity has been found in the condition of the anterior colliculi of the corpora quadrigemina or oculomotor nucleus. Pathological alteration of the entire corpora quadrigemina has been observed with intact pupils, and in other cases this region has been found to be normal, even the Edinger-Westphal nucleus, when there had been reflex rigidity of the pupils or internal ophthalmoplegia of years' duration. Reichardt thinks there is very little evidence that the cause of the reflex rigidity of the pupil is to be found in the region of the oculomotor nucleus, or even in the small cell median group of the oculomotor nucleus; indeed, contrary to such an opinion.are the cases of disease or congenital anomaly of the region of the oculomotor nucleus without reflex pupillary rigidity, and the long duration of reflex pupillary rigidity in some cases as the only ocular sign of disease and without any other manifestation of disturbance of the oculomotor nucleus. Reichardt thinks there is more reason to believe that a lesion causing reflex rigidity of the pupil is to be found in the spinal cord, and this would explain the frequent occurrence of the sign in disease of the spinal cord.

Nine cases studied clinically and pathologically by Wolff seem to show that degeneration of the posterior columns in the upper cervical region is in close relation to the rigidity of the pupils.[13] Bach, alone and in association with Meyer, has found from experiments on cats that complete even repeated division of the cervical part of the spinal cord several millimetres below the fourth ventricle causes no change of the

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[13] Wolff, Arch. für Psychiatrie, vol. xxxii, No. 1, p. 57

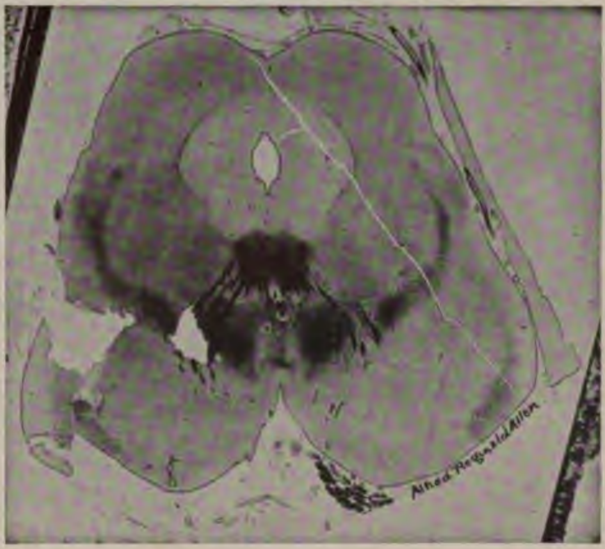


FIG. 5.—The cerebral peduncles showing the oculomotor nerves at their origin below the aqueduct of Sylvius, and their course as separate bundles of fibres to the point of exit at the inner side of the crusta. Photograph of a section from a new-born child.

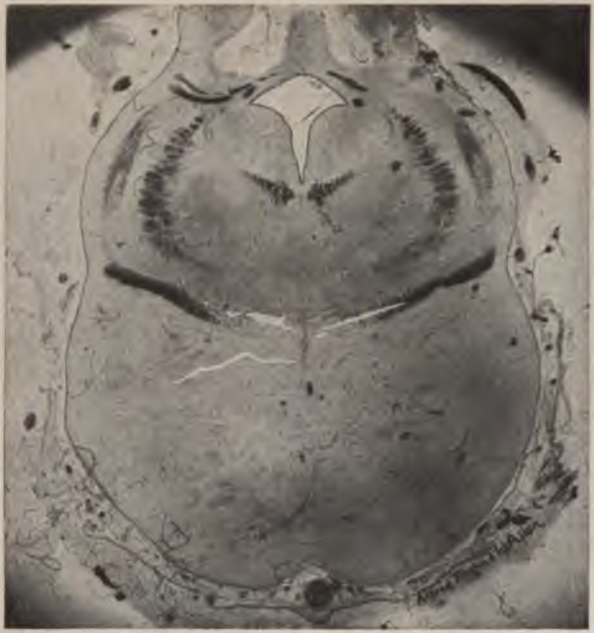


FIG. 6.—The trochlear nerves decussating above the aqueduct of Sylvius. Photograph of a section from a new-born child.

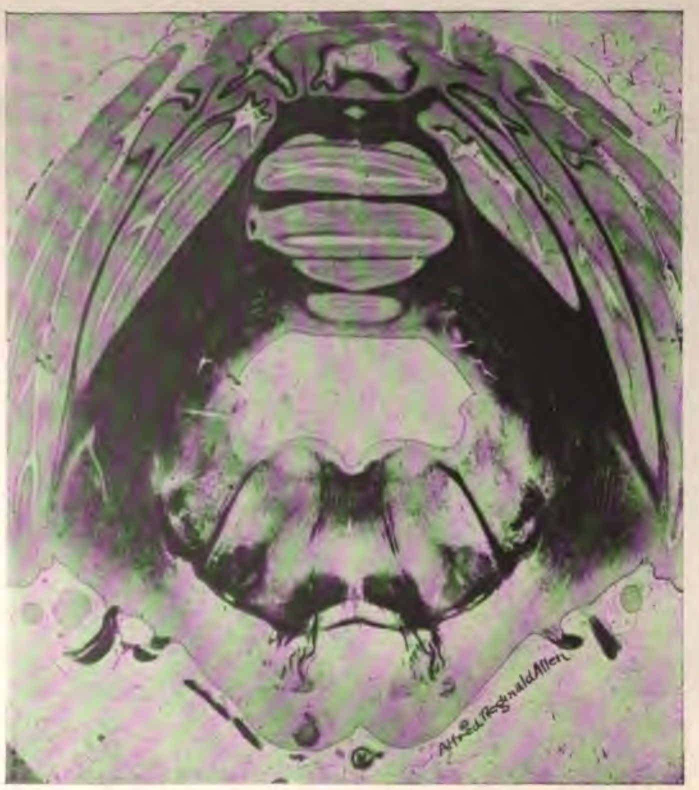


FIG. 8.—Photograph of a section of the pons from a new-born child, showing the loop formed by the fibres of the facial and abducens nerves on each side. The facial is the nerve on the outer side of the loop.

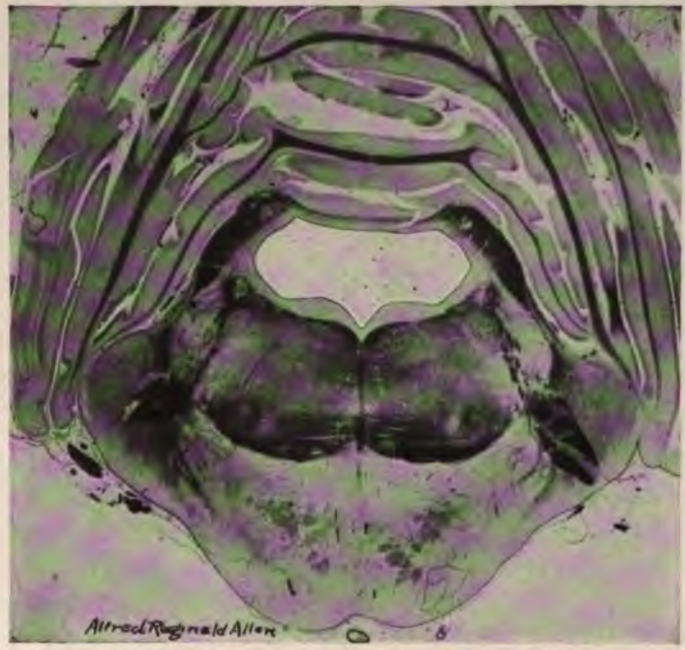


FIG. 7.—Photograph of a section of the pons from a new-born child, showing the intrapontine portions of the trigeminal nerves.

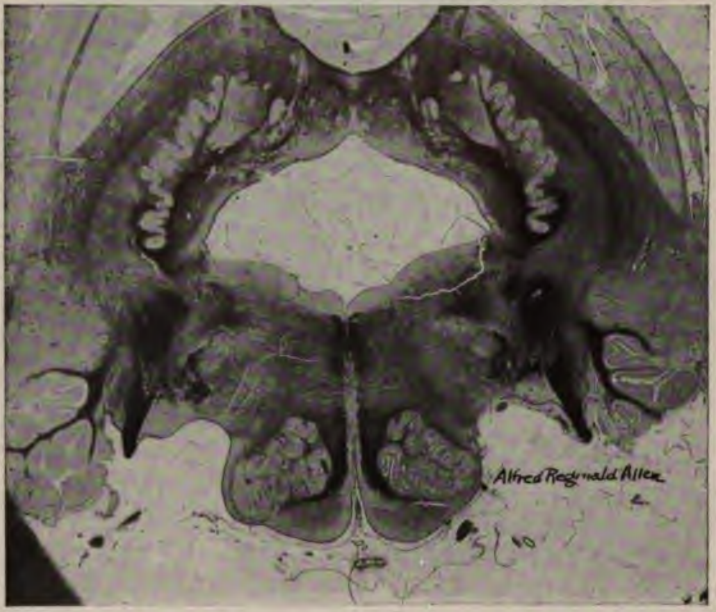


FIG. 9.—Photograph of a section from the medulla oblongata of a new-born child, showing the cochlear branch of the acoustic nerve at each side deeply stained in black.

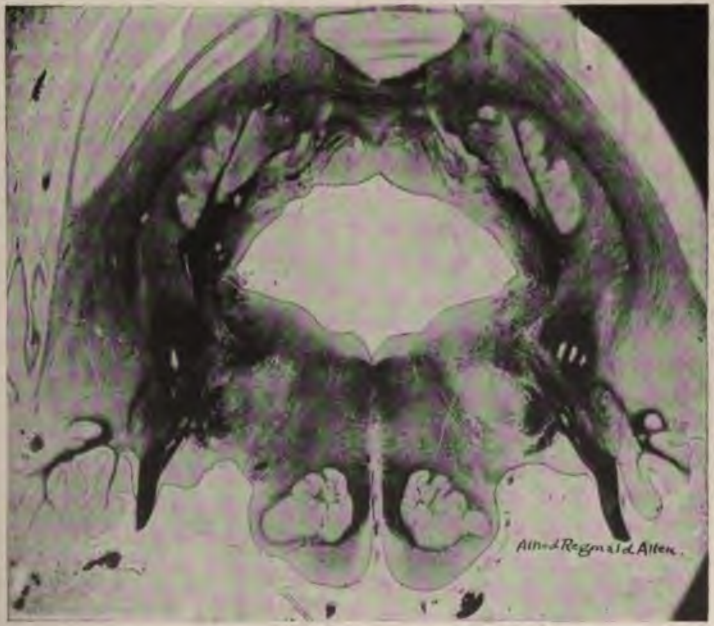


FIG. 10.—Photograph of a section from the medulla oblongata of a new-born child, showing the vestibular branch of the acoustic nerve at each side deeply stained in black.

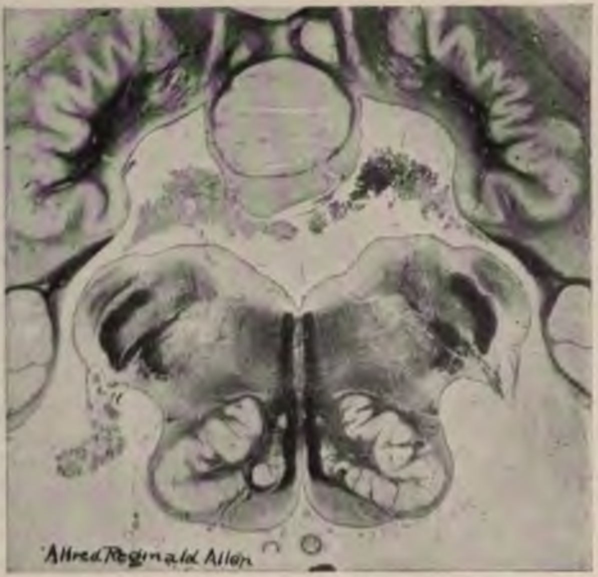


FIG. 11.—Photograph of a section from the medulla oblongata of a new-born child, showing on the right of the picture the fibres of the glossopharyngeus or vagus entering through the spinal root of the trigeminal nerve.

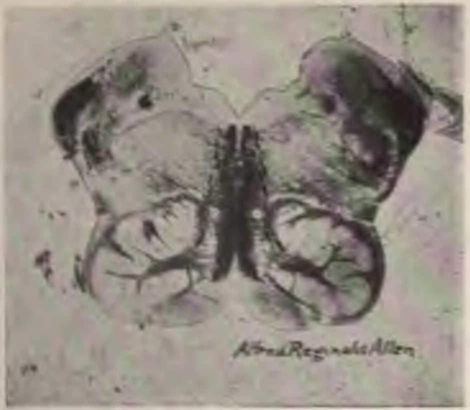


FIG. 12.—Photograph of a section from the medulla oblongata of a new-born child, showing the hypoglossus nerve on each side passing from its nucleus below the floor of the fourth ventricle and making its exit on the inner side of the inferior olive.

pupillary reaction. At the moment the division occurs the pupils become wider, but a few seconds later the light reflex is restored.

Bilateral division of the medulla oblongata at the spinal end of the fourth ventricle, at a definite place, causes rigidity of both pupils to light.

Division of the right half of the medulla oblongata at the spinal end of the fourth ventricle causes rigidity of the left pupil to light, while the reaction of the right pupil is preserved.

Mere exposure of the medulla oblongata is sufficient to diminish or even abolish the light reflex of the pupils for a quarter of an hour to one hour, and when this has occurred the rigidity has promptly disappeared when a cut has been made in the middle of the fourth ventricle or on each side in the upper part of the medulla oblongata.

These experiments seem to show that a very limited area near the median line and near the respiratory centre, at the spinal end of the fourth ventricle, is of great importance for the light reflex of the pupil. This seems to be an inhibitory centre for the pupil. Cerebral to this area is a subordinate reflex centre of the pupil. There are, therefore, probably two reflex arcs for the pupil; one passing through the corpora quadrigemina and one through the spinal end of the fourth ventricle.

TROCHLEAR NERVE.

The trochlear, or fourth, nerve arises in a distinct group of cells situated within a depression of the posterior longitudinal bundle and directly posterior to the oculomotor nucleus. It is separated from the latter group of cells. The nerve fibres on leaving the trochlear nucleus pass around the aqueduct of Sylvius on the same side, and somewhat spinalwards, decussate above the aqueduct in the anterior medullary velum, and leave the brain stem as a delicate bundle directly behind the posterior colliculus of the corpora quadrigemina of the opposite side. The nerve then descends along the outer border of the cerebral peduncle.

TRIGEMINAL NERVE.

The trigeminal, or fifth, nerve is chiefly sensory, although it has also an important motor function in the innervation of the muscles of mastication. The motor and sensory nuclei are situated side by side, the motor to the inner side, in the lateral part of the tegmentum of the pons about midway between the cerebral and spinal ends of the pons. The sensory and motor bundles of this nerve are closely associated within the pons, so that separate strands of fibres, such as are seen in the intramedullary portion of the oculomotor and abducens nerves, are not found in the course of the trigeminal nerve. The motor portion of the nerve receives a small bundle of fibres from a group of large cells situated laterally to the aqueduct of Sylvius and on the same side.

The sensory fibres arise in the Gasserian ganglion and after entering the pons terminate only partially in the sensory nucleus, the larger number of them bending downward at the level of the sensory nucleus to form the descending or spinal root, which may be traced so far as the second cervical segment of the spinal cord. Within the spinal cord it occupies a position behind the posterior horn. The long course of the descending spinal root explains the implication of the trigeminal nerve in certain cases of lesion not extending above the medulla oblongata, as in syringomyelia.

The origin of the fibres of the spinal root in the Gasserian ganglion is shown by the intense degeneration that occurs in these fibres when the sensory root of the nerve is cut between the pons and the Gasserian ganglion.

The sensory fibres on entering the pons pass dorsally and medially to the sensory nucleus. The motor root exterior to the pons remains distinct until it becomes united with the third division of the trigeminal nerve at the peripheral end of the Gasserian ganglion.

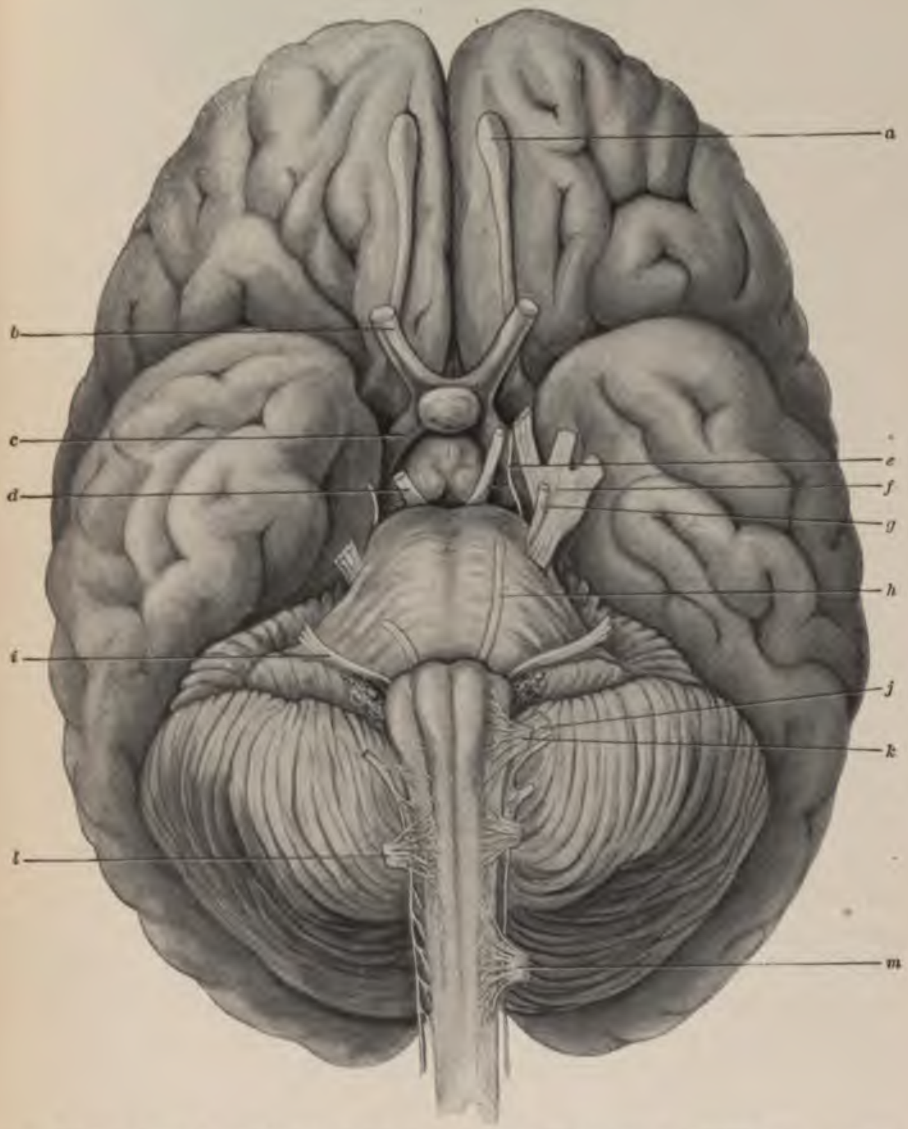
ABDUCENS NERVE.

The abducens, or sixth, nerve has its nucleus beneath the floor of the fourth ventricle in the knee made by the fibres of the facial nerve as they pass upward and then outward. The nucleus is well defined, almost spherical, and contains numerous cells. It is not probable that any of the axis cylinder processes of these cells are associated with the fibres of the facial nerve.

The fibres of the abducent nerve on leaving the dorsal and inner sides of the nucleus pass ventrally and make their exit at the junction of the pons with the medulla oblongata and near the median line. The nucleus is situated a little more cerebralward than is the point of exit of the nerve, consequently the fibres pass a little spinalward in their exit from the brain stem. The fibres are not united into one bundle in their course through the tegmentum of the pons and transversely through the pyramidal tract. They do not decussate on leaving the nucleus, although, according to some anatomists, they may receive a small addition from the nucleus of the other side.

The close association of the abducent nucleus with the fibres of the facial nerve explains the occurrence of paralysis in the distribution of these two nerves when a lesion is situated in the lower part of the

PLATE B.



a, olfactory nerve; *b*, optic nerve; *c*, optic tract; *d*, oculomotor nerve; *e*, trochlear nerve; *f*, sensory portion of trigeminal nerve; *g*, motor portion of trigeminal nerve; *h*, abducens nerve; *i*, facial and acoustic nerves; *j*, glossopharyngeal, vagus and accessorius nerves; *k*, hypoglossus nerve; *l*, first cervical root; *m*, second cervical root.

tegmentum of the pons. The nearness of the two abducent nerves to one another at their point of exit from the pons explains the occurrence of paralysis of both external rectus muscles in basal meningitis.

The abducent nucleus receives fibres from the superior olive, which is believed to be in connection with the acoustic nerve, and in this way a functional relation may be established between the two nerves. It is also in connection with that portion of the oculomotor nucleus which controls the internal rectus muscle of the opposite side. This connection is probably by means of the posterior longitudinal bundle.

At the point of exit, at the junction of the pons and medulla oblongata, the fibres of the abducent nerve become united into one bundle. From here to the exit from the skull through the sphenoidal fissure the course of the nerve is long, and consequently paralysis of the external rectus muscle is not uncommon from intracranial lesions.

FACIAL NERVE.

The facial, or seventh, nerve arises from a large group of cells situated in the upper part of the medulla oblongata at about where this structure passes into the pons. The nucleus is situated postero-laterally to the upper end of the lower olive. The fibres arising in the cells of this nucleus pass posteriorly and medially ununited toward the raphe to just below the floor of the fourth ventricle. Here the fibres ascend and bending again so as to form a knee, in which is situated the nucleus of the sixth nerve, pass ventro-laterally and on the inner side of the spinal root of the fifth nerve to their exit point at the junction of the medulla oblongata and pons. The fibres of this nerve at their exit from the pons lie on the inner side of the acoustic nerve. The loop made by the facial nerve within the lower part of the tegmentum of the pons is one of the most remarkable formations within the central nervous system, and seems to serve no definite purpose. The glossopharyngeus and vagus nerves have a similar course. From the knee onward the fibres of the facial nerve are united in a distinct bundle.

It has not been possible to distinguish a nucleus for the upper distribution of the facial nerve separate from that for the lower distribution; indeed, it seems probable that the entire facial nerve has one common nucleus in the lower part of the pons, although different branches of the nerve may be represented in different portions of the nucleus.

ACOUSTIC NERVE.

The acoustic, or eighth, nerve consists of two distinct parts, the cochlear nerve concerned with hearing and the vestibular nerve concerned with coördination. The latter enters the medulla oblongata on the inner side of the restiform body; the former passes on the outer side of this structure. The cochlear nerve extends further spinalward than does the vestibular branch, but the latter extends further cerebralward than does the cochlear branch. The fibres of the cochlear nerve terminate about the cells of the ventral nucleus and the acoustic tubercle. The ventral nucleus lies on each side of and within the substance of the cochlear nerve at its entrance into the medulla oblongata. The acoustic tubercle lies a little dorsally to the ventral nucleus, and in man is not sharply separated from it. The trapezoid body and the acoustic striæ form a part of the system connecting the cochlear nerve with structures situated higher in the brain. The former passes ventrally to the restiform body towards the raphe; the latter pass around the restiform body. The trapezoid body is much less important in man than in some of the lower animals. It is in connection with the trapezoid nucleus and superior olive, and by another system of fibres, the lateral fillet, is brought into connection with the posterior colliculus of the corpora quadrigemina.

The vestibular nerve enters the medulla oblongata between the restiform body and the descending spinal root of the trigeminal nerve. The fibres of this nerve terminate about the cells of the posterior nucleus below the floor of the fourth ventricle, and in the nuclei of Deiters and von Bechterew, below and lateral to the floor of the fourth ventricle. The vestibular nerve possesses also a descending root, between the fibres of which are numerous nerve-cells.

GLOSSOPHARYNGEAL AND VAGUS NERVES.

The glossopharyngeus, the vagus, and the cerebral portion of the accessorius cannot be sharply separated at their exit from the medulla oblongata. The fibres of these nerves are found directly dorsal to the lower olive and in the continuation downward of this line of exit. The uppermost fibres belong to the glossopharyngeus; the middle to the vagus, and the lowest to the accessorius. The fibres are not united into three nerve bundles at their point of exit from the medulla oblongata.

The sensory glossopharyngeus and sensory vagus nerve-fibres enter the medulla oblongata laterally, passing through the spinal root of the trigeminal nerve. Some of these terminate in the dorsal nucleus below the floor of the fourth ventricle and lateral to the hypoglossus nucleus. Others bend downward near the lateral part of the fourth ventricle to form the descending root. This root is accompanied by a small amount of gray matter, and about the nerve-cells of this many of the fibres of the descending root terminate. In the opinion of some anatomists, the descending root contains chiefly glossopharyngeus fibres and few vagus fibres.

The posterior nucleus is regarded by some anatomists as motor in function and not sensory. The ventral nucleus, or nucleus ambiguus, is in all probability motor. This nucleus is not sharply defined. It lies dorso-laterally to the lower olive. In some transverse sections only a few large cells belonging to this nucleus may be seen; in other sections no cells at all are found. The nerve-fibres arising in this nucleus pass separately dorsally and medially, much in the same way as do the fibres of the facial nerve, but before reaching the fourth ventricle bend to form a knee, and join the sensory fibres passing obliquely through the medulla oblongata.

ACCESSORIUS NERVE.

The accessorius, or eleventh, nerve has been described so far as its cerebral portion is concerned in connection with the glossopharyngeus and the vagus. The spinal portion arises in the lateral horn of the spinal cord, and the nerve-fibres passing from the cells in this situation reach the periphery of the cord through the lateral column. The nerve-fibres can be observed leaving the cord as separate strands as low as the fifth or sixth cervical segment.

The upper fibres, or cerebral portion, of the accessorius probably belong to the vagus nerve.

HYPOGLOSSAL NERVE.

The hypoglossus, or twelfth, nerve has its nucleus just below the floor of the fourth ventricle close to the raphe. It is a well defined group of large nerve-cells, resembling closely those of the anterior horns of the spinal cord. The nerve-fibres arising in these cells pass a little obliquely forward in straight lines until they reach the lower olive, but they are more or less diverted from their course in passing through this structure. They leave the medulla oblongata lateral to the pyramid, although some of the nerve-fibres pass directly through the pyramid.

The nucleus of the hypoglossus is one of the easiest to find of all the motor nuclei in the medulla oblongata and pons, and is about 18 millimetres in length, extending upward about as far as the striæ medullares.

CHAPTER II.

SIGHT: THE STRUCTURE AND OPTICAL PROPERTIES OF THE EYE.

BY WILLIAM NORWOOD SOUTER, M.D.

SIGHT, or vision, is that sense by which we appreciate the form and color of objects through the action of the physical energy, *light*, on the retina. In other words, sight is the consciousness which results from stimulation of the retina by light. The visual apparatus, therefore, consists of two distinct parts: (1) the eye, which is comparable to a photograph camera, the retina (which receives and transforms the light-energy into a nerve-impulse) corresponding to the sensitized plate of the camera; and (2) the conducting and interpreting mechanism, by means of which the nerve-impulse is conducted to the visual areas of the brain and thence to the centres of consciousness where the impulse is manifested as sight.

The manner in which the light-stimulus is transformed into a nerve-impulse is but little understood; it is only known that in this transformation a certain chemical change (destruction of the retinal purple) takes place in the retina. The second transformation, that of the nerve-impulse into the consciousness of vision, is also beyond our comprehension. So much as is known of the mental process of vision will be considered in a subsequent chapter; it is the province of the writer of the present chapter to consider only the structure of the eye, and its adaptation and imperfections as an optical mechanism. (See chapter III.)

THE STRUCTURE OF THE EYE.

The eyeball is spheroidal in shape. Its normal diameter varies between 23 millimetres and 25 millimetres. The antero-posterior diameter is slightly greater than the transverse diameter, while the vertical is the least of the three dimensions. The difference between the greatest and the least diameter is about 1 millimetre.

As externally viewed the eye is divisible into two distinct portions: an anterior, transparent portion, or *cornea*, and a posterior, larger, opaque portion, the *sclera*. The curvature of the cornea is greater than that of the sclera, so that at the junction of these two structures there is formed a groove or sulcus—the *sulcus corneæ*. On antero-posterior meridional section of the eye the corneal portion comprises about one-sixth and the scleral portion the remaining five-sixths of the circumference.

The eyeball is protected posteriorly by the bony walls of the orbit, but antero-exteriorly there is no other protection than that afforded by the lids. The latter serve, when partially or completely closed, to protect the eye from excessive light and to some extent from injury by foreign bodies. The inner surface of the lids is covered with mucous membrane, the *conjunctiva*, which is reflected at the upper and lower fornices (the upper and lower culs-de-sac) upon the eyeball, and the epithelium of this membrane is continued over the anterior surface of the cornea. The conjunctiva thus forms a sac, open anteriorly at the lid-margins.

There are three concentric coats or tunics of the eye: the external, the middle, and the inner coat.

The External Coat.—The external coat consists of the transparent cornea anteriorly and the opaque sclera posteriorly. The structure of these two membranes is essentially the same, both being of a fibrous nature. The transparency of the cornea is the result of close union and regular arrangement of the fibres, and of homogeneity of refractive index of the component corneal tissues.

The Cornea is covered anteriorly by *epithelium*, which is continuous with the epithelium of the conjunctiva. Immediately behind the epithelium is the *anterior limiting membrane* (Bowman's membrane), which is a dense, structureless membrane. Next to this is the corneal stroma, which consists of regularly arranged lamellæ of fibrous tissue having lymph spaces or *lacunæ* connected by small canals or *canaliculi.* In each lacuna there is situated a cell whose processes extend along the canaliculi so as to communicate with adjacent cells. These are the *fixed cells* of the cornea, in contradistinction to the leucocytes or *movable cells* which are carried along the lymph channels. The fourth layer of the cornea is a thin, homogeneous, very resistant membrane, known as *Descemet's membrane.* It is continuous with the ligamentum pectinatum (p. 23), which, together with Descemet's membrane, is embryologically a part of the middle coat of the eye. The fifth and innermost layer is the *endothelium*, or layer of polygonal cells, which is continuous with the endothelium of the iris, and which, like the preceding layer, is embryologically a part of the middle coat. There are no *blood-vessels* in the healthy cornea, nourishment being supplied through lymph channels.

The corneal nerves are derived from the ciliary plexus. They penetrate the sclera on the outer side of Schlemm's canal and form a network in the vicinity of the corneal margin. From this plexus branches are given off which supply the several layers of the cornea.

The radius of curvature of the anterior surface of the cornea is about 8 millimetres, 7.8 millimetres being the commonly accepted average for the normal eye. The radius of curvature of the posterior surface is about 6 millimetres. The thickness of the cornea at its centre is about 1 millimetre.

The sclera is, as previously stated, composed of dense fibrous tissue irregularly arranged. It is of a glistening whitish color, which indicates its scant blood supply. The sclera is pierced by a number of openings for blood-vessels and nerves. The anterior ciliary arteries, veins, and nerves enter the eyeball near the margin of the cornea. In the equatorial region there are four or five openings for the passage of the venæ vorticosæ. Posteriorly there are a number of small openings for the posterior ciliary arteries and veins, and a large opening for the optic nerve. This opening is about 1.5 millimetres in diameter, and is situated slightly to the nasal side of the posterior pole of the eye; it is traversed by connective-tissue fibres, continuous with the inner layers of the sclera, which are known as the *lamina cribrosa*. The outer layers of the sclera are continuous with the sheath of the optic nerve.

The greatest thickness of the sclera is about 1 millimetre, near the posterior pole. From this region it becomes somewhat thinner toward the equator, and is again thickened anteriorly where the tendons of the extraocular muscles blend with the sclera.

Anteriorly, near the sclero-corneal junction and concentric with it is found the circular sinus, known as *Schlemm's canal*. The nature of this was for a long time in doubt, but it is now regarded as a venous channel, into which drains also the aqueous humor through the ligamentum pectinatum.

The sclera is covered externally by a thin layer of loose connective tissue, the *episclera*, which is more freely supplied with blood-vessels than the sclera proper.

The anterior portion of the sclera is covered by conjunctiva which is reflected from the lids at the upper and lower culs-de-sac; the posterior portion is embedded in Tenon's capsule, from which it is separated by a lymph space, thus allowing the eye to be freely movable in the capsule.

The Uvea.—The middle coat or uvea is so named from its resemblance (when stripped of the sclera) to a grape.

The iris or anterior portion of the uvea is a diaphragm having in it a circular opening (the pupil) for the passage of light to the retina.

From the free pupillary border the iris extends peripherally to the ciliary body, to which it is attached slightly behind the sclero-corneal junction. The angle between the sclero-cornea and the root of the iris is bridged over by a loose, spongy tissue, called the *ligamentum pectinatum.* The patulency of this structure is of great importance in the regulation of intraocular tension, since through its meshes the aqueous humor is drained off into Schlemm's canal, of which the ligamentum pectinatum forms the inner wall.

The iris is composed of an *endothelial layer* in front (continuous with the endothelium of the cornea), a *pigment layer* behind (continuous with the pigment layer of the retina), and, between these two, the *stroma.* The latter is a network of blood-vessels and fibrous tissue, in which are embedded the two muscles of the iris, the *sphincter* and the *dilator.* The former is a circular band of muscular tissue about 1 millimetre in width, which surrounds the pupil and causes contraction of this opening. The dilator muscle consists of longitudinal or radial fibres which assist in dilating the pupil. These two muscles regulate the size of the pupil, which contracts in brilliant illumination to cut off the excess of light, and expands in feeble illumination to allow sufficient light to enter the eye.

The bluish color of the iris in blondes is due to the pigment contained in the posterior or retinal pigment layer; in dark eyes there is additional pigment in the stroma.

The ciliary body, the middle portion of the uvea, is ring-shaped, triangular on transverse section, with its broadest side or base in apposition with the inner surface of the sclera (Fig. 1). This body extends from the sclero-corneal junction backward for a distance of about 2 millimetres, being merged in the chorioid at the ora serrata, or anterior extremity of the retina. The ciliary body is composed of the ciliary processes and the ciliary muscle. The *ciliary processes* are connective-tissue elements in which are embedded a large number of blood-vessels and pigment-cells. There are from fifty to seventy of these processes longitudinally arranged with their larger ends projecting anteriorly. The *ciliary muscle* lies beneath the processes and consists of two parts. The larger part is formed of meridional fibres (*Brücke's muscle*) which are attached anteriorly with firm union to the sclero-corneal junction and neighboring sclera. The fibres of this part pass backward and are inserted into the anterior portion of the chorioid. This is the most external part of the muscle. To its inner side and adjoining the processes is the second or transverse part, the *annular muscle of Müller,* which consists of a band of muscular fibres surrounding the periphery of the iris. This part constitutes about one-tenth of the entire muscle in the normal or emmetropic eye. In hyperopia the annular muscle is larger and in myopia it is smaller than in emmetropia. The ciliary muscle is an essential factor in the accomplishment of accommodation. By contraction of this muscle the crystalline lens becomes more convex, so as to enable near objects to be seen clearly.

The chorioid is that part of the uvea which is posterior to the ciliary body. It is dark brown in color, thus differing from the ciliary body, which is black; its inner surface is smooth, while the outer surface when torn from the sclera presents a shaggy appearance. In this respect and also in the abundance of blood-vessels the chorioid resembles the embryonic chorion, from which the name is derived. The thickness of the chorioid varies from .08 millimetre near the optic nerve to .01 millimetre at the ora serrata.

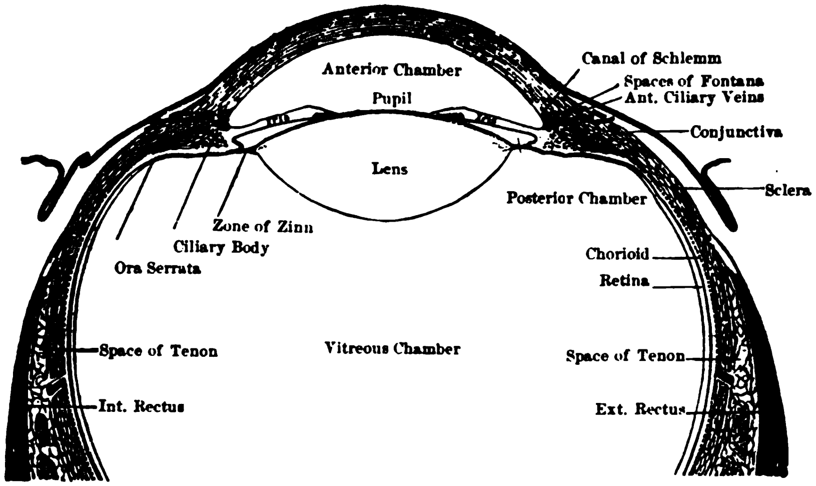


FIG. 1.—Diagram of anterior segment of eye, drawn to accurate scale. (Flemming.)

In structure the chorioid consists of a network of blood-vessels, intervascular connective tissue, and pigment. It is usually described as consisting of five layers, which, beginning with the external layer, are: (1) The supra-chorioid, (2) the layer of large vessels, (3) the layer of medium vessels, (4) the layer of capillaries, and (5) the lamina vitrea.

The *supra-chorioid* is composed of non-vascular but richly pigmented lamellæ, which lie between the sclera and the chorioid proper. When stripped from the sclera these lamellæ are torn in shreds, from which the chorioid acquires its shaggy appearance.

The layer of large vessels, consisting chiefly of veins, is placed in a close network, with many anastomoses. The intervascular spaces are highly pigmented.

The layer of medium-sized vessels is thin and only slightly pigmented.

The capillary layer consists of large capillaries, closely arranged. This layer contains no pigment.

The lamina vitrea is a thin homogeneous membrane which coats the inner surface of the capillary layer.

The blood supply of the uvea is derived from the anterior and the posterior ciliary arteries. The anterior ciliary arteries, four in number, arise from the four arteries supplying the recti muscles, pierce the sclera near the cornea, and terminate in the greater arterial circle of the iris. From this circle radial branches are given off to supply the iris, and these, by reunion, form the lesser arterial circle near the pupillary border of the iris. The posterior ciliary arteries consist of the long and the short ciliary arteries. Of the former there are two, which arise from the ophthalmic artery, pierce the sclera near the optic nerve, and continue forward, one on each side of the eyeball, between the sclera and chorioid as far as the ciliary muscle. Each artery then divides into two branches, which form, with the aid of the anterior ciliary arteries already mentioned, the greater arterial circle of the iris. It thus appears that the anterior and the long posterior ciliary arteries supply chiefly the iris and ciliary body. The short posterior ciliary arteries, from twelve to fifteen in number, arise from the ophthalmic or some of its branches, and surround the optic nerve as they pass to the posterior part of the eyeball, where they pierce the sclera and supply the chorioid and ciliary processes.

The greater part of the venous blood from the uvea leaves the eye through the venæ vorticosæ, of which there are from four to six penetrating the sclera in the equatorial region of the eyeball. A much smaller proportion of the venous blood, chiefly that from the ciliary muscle, leaves the eye through the anterior ciliary veins, which pierce the sclera near the cornea and pass backward under the conjunctiva. These veins anastomose with the conjunctival veins and also with Schlemm's canal.

The nerves of the uvea are the long and the short ciliary nerves. The former, two or three in number, are derived from the nasal branch of the fifth nerve. The short ciliary nerves, from ten to twelve in number, are derived from the ciliary ganglion; they contain motor fibres from the third nerve, sensory fibres from the fifth nerve, and fibres from the sympathetic system.

The Retina.—The retina is the third or innermost eye-tunic. It is formed by the terminal expansion of the optic nerve, and, with the exception of its outer or pigment layer, is transparent during life, having only a slight purplish tinge (*the retinal purple*), which fades on exposure to light.

The retina extends forward as far as the ora serrata, beyond which it exists only in rudimentary form. In the latter condition it extends to the border of the pupil, furnishing the posterior pigment layer of the ciliary body and iris (*pars ciliaris retinæ* and *pars iridica retinæ*), which is continuous with the pigment layer of the retina proper (*pars optica*). This pigment layer is embryologically a part of the retina, but it adheres to the chorioid when the retina is lifted from the former membrane.

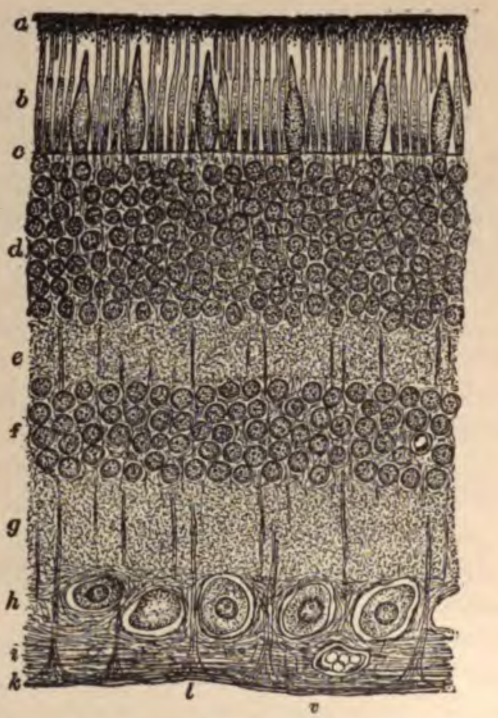


FIG. 2.—Section of human retina, showing the usual appearance of the component layers in ordinary preparations.—*a*, pigment layer; *b*, rods and cones; *c*, external limiting membrane; *d*, outer nuclear layer; *e*, outer plexiform layer; *f*, inner nuclear layer; *g*, inner plexiform layer; *h*, layer of ganglion-cells; *i*, fibre layer; *k*, internal limiting membrane; *l*, base of Miller's fibre; *v*, blood-vessel. Magnified 475 diameters.

The fibres of the optic nerve, which within the eye consist only of axis-cylinders (except in the anomalous condition known as opaque nerve-fibres), radiate from the *optic disk*, or entrance of the optic nerve, and form the inner layer of the retina. The optic disk is situated slightly to the nasal side of the posterior pole of the eye. At the posterior polar region is situated the *macula lutêa* or *yellow spot*. This is described as an annular or oval area from 1 millimetre to 2 millimetres in diameter, at the centre of which lies a minute depression, the *fovea centralis*.

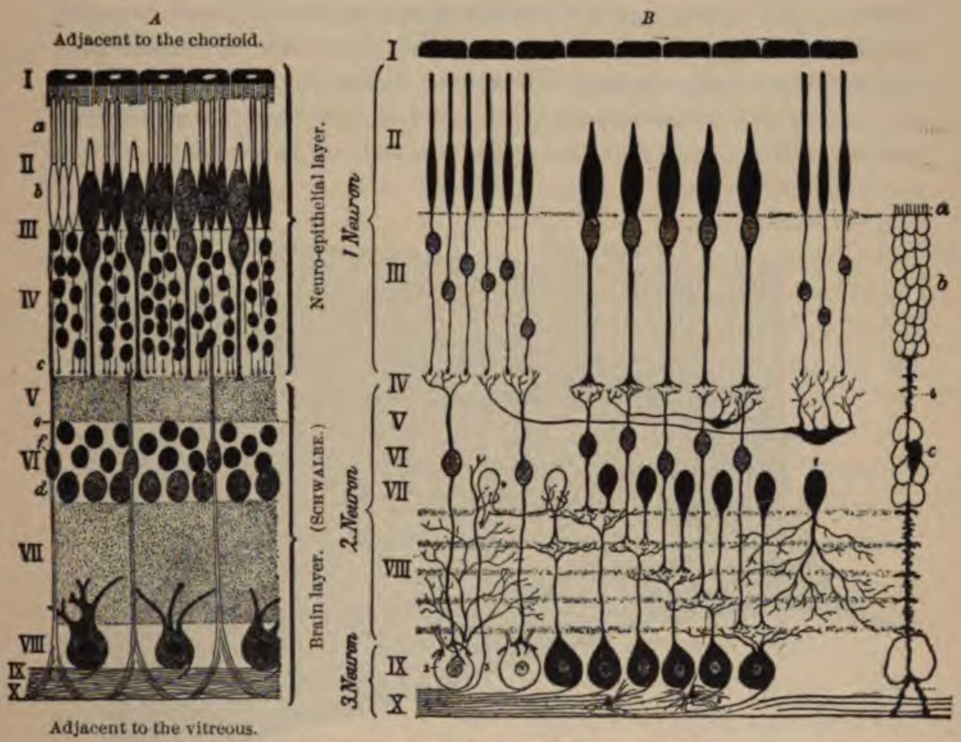


FIG. 3.—Scheme of the structure of the human retina.

A. Horizontal section, hæmatoxylin stain. I. Pigment epithelial layer. II. Layer of rods and cones: *a*, external; *b*, internal elements. III. External limiting membrane. IV. External molecular layer: *c*, fibre layer. V. External granular layer. VI. Internal molecular layer: *d*, spongioblasts; *e*, supporting fibres of Müller; *f*, nuclei of the same. VII. Internal granular layer. VIII. Layer of ganglion cells. IX. Nerve-fibre layer. X. Internal limiting membrane.

B. Demonstration after the method of Golgi. I. Pigment epithelial layer. II. Layer of rods and cones. III. Molecular and visual cells. IV. External plexiform layer. V. Layer of horizontal cells. VI. Layer of bipolar cells. VII. Layer of amacrine cells. VIII. Internal plexiform layer (fibre layers). IX. Layer of ganglion cells. X. Nerve-fibre layer: 1, diffuse amacrine cells; 2, diffuse ganglion cells; 3, centrifugal nerve fibres; 4, association-amacrine cells; 5, neuroglia cells; 6, supporting fibres of Müller.

In structure the retina is composed of an outer and an inner lamina. The outer lamina constitutes the retinal pigment layer. The inner lamina constitutes the rest of the fully formed retina (Fig. 2). The inner lamina is subdivided into two layers, the neuro-epithelial and the cerebral (Fig. 3). The *neuro-epithelial layer* consists of the retinal rods and cones, which constitute the light-receiving and transforming mechanism. The *cerebral layer*, which is the impulse-transmitting part of the retina, contains a layer of bipolar cells and a layer of ganglion-cells and nerve-fibres. The bipolar cells connect by their processes the rods and cones on the one hand with the ganglion-cells of the nerve-fibres on the other, and thus form the connecting link between the peripheral recipient elements and the cerebral conducting nerve-fibres. The bipolar cells are not united to the rods and cones or to the ganglion-cells, but their process-terminals are in close apposition with the terminals of the elements which they connect.

In addition to the bipolar cells just described there are also found similar cells, except that their processes extend horizontally. From their apparent nerve connections these cells are called *association cells,* though their function is not definitely known. Still other cells are found which apparently have no nerve connections, and whose function is likewise not known. They are called *spongioblasts* (Müller) or *amacrine cells* (Cajal).

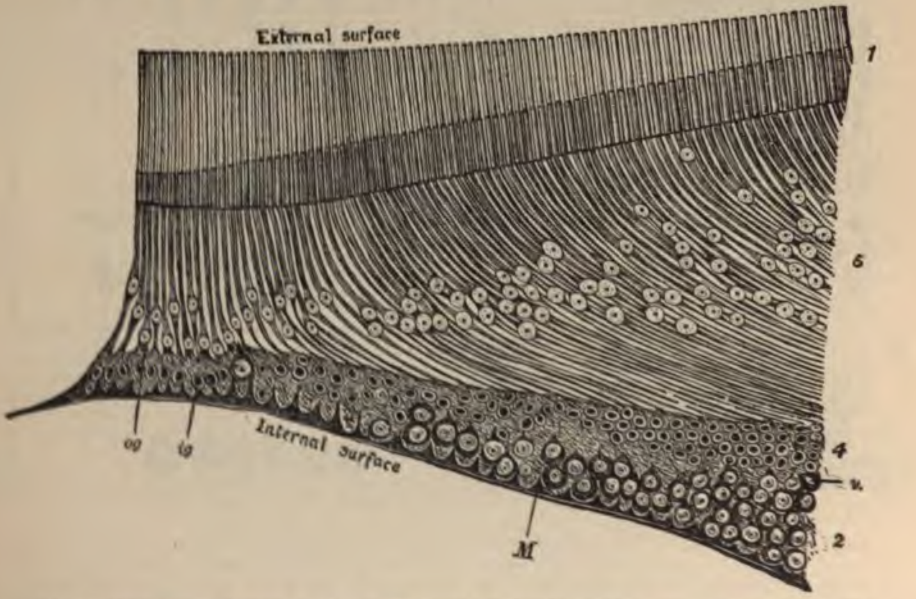


Fig. 4.—Diagrammatic section of the human fovea. Magnified 375 diameters. (Golding-Bird and Schäfer.)—2, ganglion layer; 4, inner nuclear layer; 6, outer nuclear layer, the cone-fibres forming the so-called external fibrous layer of Henle; 7, cones; v, section of a blood-vessel; *M*, membrana limitans interna; *og*, *ig*, outer and inner granules (cone-nuclei and bipolars) at the centre.

The supporting neuroglia or *sustentacular tissue of Müller* consists of transverse fibres and an outer and an inner membrane, the *external* and the *internal limiting membrane*, respectively. These two membranes, together with the retinal pigment layer and the various subdivisions of the neuro-epithelial and cerebral layers, make up the ten layers into which the retina is usually divided.

The macula lutea, that part of the retina which is concerned in distinct vision, requires special description. In this region the rod-elements are replaced by cones, so that well within the border of the macula there are no rods, while the number of cones is very great. The layer of ganglion-cells is also much thickened in this area, except at the fovea, the region of greatest visual acuteness, where the ganglion-cell layer becomes very thin; at the foveal centre these cells are not found, the connection between the cones and the nerve-fibres being made by laterally extending processes of the bipolar cells. Transversely the processes of these cells are much shorter at the fovea than elsewhere. This fact, together with the diminution of the ganglion-cells, makes the cone-elements the most prominent feature at the fovea. The cone-fibres and nuclei, constituting the outer nuclear layer (IV, Fig. 3, A) are relatively conspicuous; they form the *external fibrous layer of Henle.* The fibres are situated obliquely at the sides of the fovea, but perpendicularly at its centre (Fig. 4). In the latter situation the fibres are much shorter than at the sides, thus adding another factor in the causation of the depression at the fovea; the principal other factors are the thinning of the nerve-fibre layer (ganglion-cells) and the sessile character of the bipolar cells.

Blood Supply of the Retina.—The outer layers of the retina receive their blood supply from the capillaries of the adjacent chorioid. The inner layers are supplied by the central artery of the retina. This artery is derived from the ophthalmic artery; it penetrates the optic nerve about 5 millimetres behind the eyeball and emerges at the optic disk, slightly on the nasal side of its centre. It is divided at its point of emergence or slightly behind the disk into two branches, an upper and a lower branch. These branches traverse the disk giving off small branches, and again divide just within or without the border of the disk into temporal and nasal branches. The further subdivisions are illustrated in Fig. 5. The branches of the retinal artery are terminal, that is, there are no anastomoses, and consequently embolism of a branch is followed by loss of function.

The efferent veins, slightly larger than the arteries, follow the same general course as the arteries. Although there are no large vessels at the macula, this region is abundantly supplied with capillaries, except at the fovea, where there are no blood-vessels.

Refractive and Distensive Contents of the Eye.—Within the inner tunic of the eye just described are included the aqueous humor, the crystalline lens, and the vitreous body. These substances serve the double purpose of keeping the otherwise collapsible eyeball distended and at the same time of acting as refractive media.

The aqueous humor is a transparent, slightly alkaline fluid, composed chiefly of water and sodium chloride; it is secreted by the ciliary body, passes forward through the pupil (Fig. 1), and is carried off through the interspaces of the ligamentum pectinatum into Schlemm's canal.

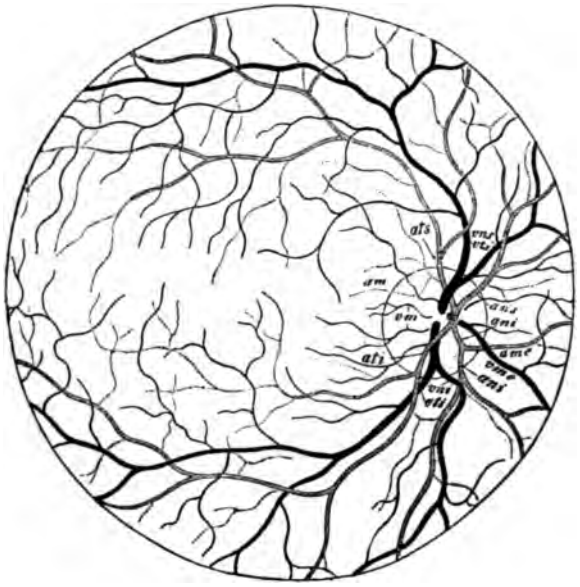


Fig. 5.—Diagram of the blood-vessels of the human retina. (Leber, after Jaeger.)—*ans*, *vns*, superior nasal artery and vein; *ats*, *vts*, superior temporal artery and vein; *ani*, *vni*, inferior nasal vessels; *ati*, *vti*, inferior temporal vessels; *ame*, *vme*, median vessels; *am*, *vm*, macular vessels.

The crystalline lens is formed of delicate fibres closely united. In childhood the lens substance is gelatinous. In early adult life the central part or nucleus is solid, while the outer or cortical part is soft. In old age the entire lens is solid. The fibres are arranged in regular layers, and the lens is divisible into three principal segments. With the diminution of transparency which takes place in old age, the lines of union of these segments become visible when the lens is examined with the aid of oblique illumination.

The lens is enclosed in a delicate capsule, to which is attached, anteriorly and posteriorly, near the periphery or equator of the lens, the suspensory ligament (*zonule of Zinn*). By this ligament, the outer border of which is attached to the ciliary body, the lens is held in position (Fig. 1).

In shape the lens is biconvex, its anterior surface being less curved than its posterior surface. The radius of curvature of the anterior surface is about 10 millimetres, while that of the posterior surface is about 6 millimetres. In childhood, and to a less degree at a later age, prior to complete solidification, the curvature of the anterior surface of the lens is capable of increase by contraction of the ciliary muscle and consequent relaxation of the suspensory ligament. In this way the eye is adapted for different distances as is required in near vision.

The lens is a bloodless body after birth, and its metabolism is extremely slow, especially in its inner or nuclear portion. It is probable that as much nutritive fluid as is required is supplied from the adjacent ciliary body.

The vitreous body, which occupies the larger and posterior part of the eye, consists of a transparent, semi-fluid, colloid substance, together with trabeculæ of supporting tissue. The whole is enclosed in a capsule, the hyaloid membrane, on the anterior, concave surface of which the crystalline lens rests. The vitreous also contains floating cells (*muscæ volitantes*) which can usually be seen entoptically when one looks at a uniformly bright light, as the sky. In disease these cells sometimes become greatly multiplied, so as seriously to interfere with vision.

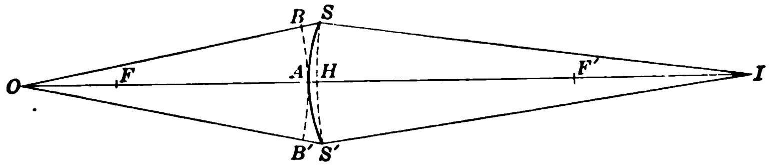
Like the lens, the vitreous is devoid of blood after birth, and consequently must receive its nourishment from surrounding tissues, the ciliary body and chorioid. There is also a central canal (which lodges the pre-natal hyaloid artery) running from the posterior pole of the lens to the optic disk and communicating with the lymph spaces of the optic nerve. This canal doubtless plays an important part in the metabolism of the vitreous.

THE EYE AS A REFRACTIVE APPARATUS.

In order that we may perceive the form of an object by the visual sense the stimulated area on the retina must constitute an image of that object. This image is formed by the refracting apparatus of the eye. It is well known that light appears to travel in straight lines (*rays*) as long as the medium is unchanged, but when light passes from one medium to another of different density or refractive index the path of all rays which meet the separating surface obliquely undergoes a change of direction. This change in direction is called *refraction;* it is explained by assuming that light travels in waves and that the progress of these waves is less rapid in dense than in rare substances.

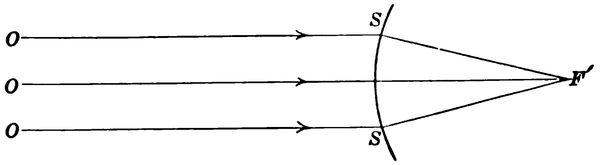
By means of a properly curved surface separating two media a considerable portion (*a pencil*) of the rays proceeding from a point may be united in another point or focus after refraction, as is illustrated in Fig. 6, in which *SAS′* represents a section of a spherical surface separating two media, the second medium (that on the right of the figure) being denser than the first, while *SOS′* represents a section of a pencil of light proceeding from *O*. That portion of the wave which travels along *OA* encounters the denser substance sooner than the portions which travel along *OS* and *OS′*. While the first portion travels the distance from *A* to *H* in the denser substance, the peripheral portions travel the greater distances *BS* and *B′S′* in the rarer medium. When the whole pencil has entered the denser medium *SHS′* represents the form of the wave front, and *SI, OI,* and *S′I* represent rays (perpendicular to the wave front), all meeting in the *focus, I.* The ray *OI,* which meets the separating surface perpendicularly and which consequently undergoes no refraction, is called the *optic axis.*

FIG. 6.



The position of the focus *I* depends upon the degree of refraction,—that is, upon the curvature of the surface and the relative densities (refractive indices) of the two media. For the same surface and media the position of the focus also varies with the point of origin of the incident pencil. As the point *O* becomes more distant from the surface the focus *I* approaches the surface, and *vice versa.* The two points *O* and *I* are called *conjugate foci.* When the anterior focus *O* is so far distant that the wave front may be regarded as plane and the rays as

FIG. 7.



parallel, the conjugate focus is called the *posterior principal focus* (Fig. 7). When, on the other hand, the point *O* approaches so near the surface that the rays are parallel after refraction, that is, when the conjugate *I* is infinitely distant, the point from which the rays proceed is called the *anterior principal focus* (Fig. 8). When the point of origin is nearer than the first principal focus the rays will remain divergent after refraction, but the divergence will be less than before refraction. In this case the rays appear to proceed from a point, *I*, on the left of *O* (Fig. 9), but since they do not actually pass through this apparent point of origin, this is not an illuminated point as is the focus *I* in Fig. 6. Hence the latter is called a *real* focus, while the former is a *virtual* focus. So also, if the rays have been rendered convergent by previous refraction, so that they are already converging toward a focus, the convergence of the rays will be increased by the refraction at the convex surface (provided the focus toward which the rays converge is beyond the centre of the surface), so that after this refraction the rays will meet in a real focus which is nearer the surface than the principal focus. This condition is represented in the correction of hyperopia by a convex lens, as is illustrated in Fig. 14, p. 41. The path of the light motion may be reversed in any of the preceding diagrams; that is, these diagrams may be used to illustrate the refraction which takes place when light passes from a *dense* to a *rare* medium at a *concave* surface. On the other hand, it is apparent that the refraction which takes place when light passes from a *rare* to a *dense* medium at a *concave* surface or from a *dense* to a *rare* medium at a *convex* surface is

FIG. 8.

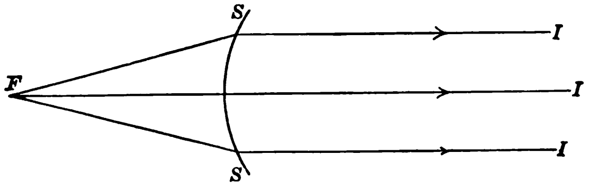
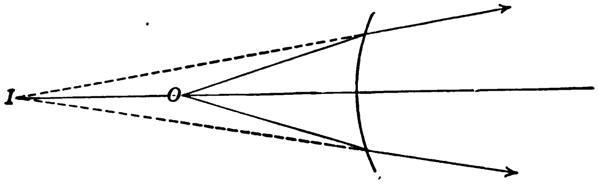


FIG. 9.

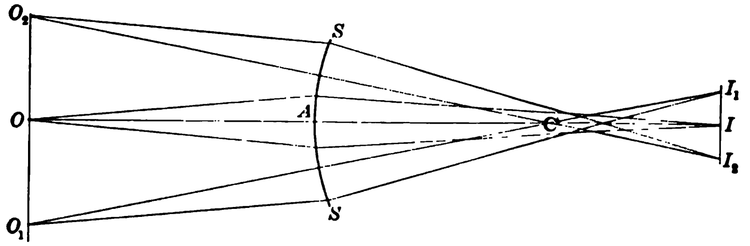


opposite to that hitherto described; that is, such refraction has the effect of increasing the divergence or diminishing the convergence of rays.

As the various rays proceeding from O (Fig. 10) are concentrated at I, so the rays from O_1 are focused at I_1, and those from O_2 are focused at I_2. Hence I_1I_2 is the image of O_1O_2. All the rays which pass through the centre C meet the surface perpendicularly and consequently are unrefracted. The point C is called the *nodal-point* and the unrefracted rays are called nodal-rays, or optic axes, OI being the ***principal axis*** and O_1I_1 and O_2I_2 *secondary axes.*

Since O_1O_2C and I_1I_2C are similar triangles, we know that the linear dimensions O_1O_2 and I_1I_2 are proportional to the distances OC and IC, or each linear dimension of the image bears the same proportion to the corresponding dimension of the object as does the distance of the image from the nodal-point to the distance of the object from this point. Hence it is apparent that the nearer an object is to the refracting surface (as the eye) the larger will be the image as formed after refraction.

FIG. 10.



The cornea of the eye presents a curved (approximately spherical) surface, convex to incident light, and the refractive index of the cornea and aqueous humor is greater than that of air;[1] hence the condition of refraction when light enters the cornea is similar to that represented in Fig. 10. The corneal refraction, however, is not sufficiently great to bring even parallel rays to a focus on the retina of the normal eye, and still less would it be able to focus rays from a near object. The deficiency is supplied by the crystalline lens, whose refractive index is higher than that of the aqueous and vitreous, and which is biconvex in form. By the additional convergence given rays of light in passing through this lens they are brought to a focus on the retina in the normal eye, and consequently a well-defined image of the object of vision is formed on the retina.

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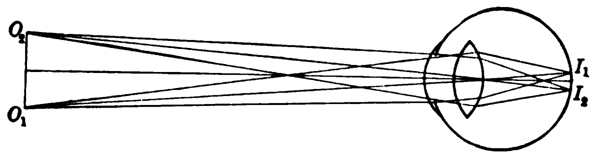
[1] The refractive index of the aqueous is practically identical with that of the vitreous, while the index of the cornea is slightly greater than that of the aqueous and vitreous; but for the sake of simplicity a common index (1.337) may be assumed for these three media.

A combination of several surfaces and media, such as is formed by the cornea, aqueous, lens, and vitreous, constitutes a *compound optical system.* Such a system is analogous to a simple system of one surface and two media, except that, instead of both conjugate focal distances being measured from the same point (where the surface and principal axis intersect), the anterior and posterior focal distances are measured respectively from the *first* and the *second principal point,* and instead of a single nodal-point there are two *nodal-points,* separated by the same interval as the principal points. In a compound system the nodal-ray does not form a continuous straight line, but its characteristic is that after the last refraction its direction is parallel to that which it had before entering the refracting system. In the eye the lateral displacement of the nodal-ray is so slight, the two nodal-points being separated by an interval of only .37 millimetre, that it would escape observation in a diagrammatic drawing of the natural size. For practical purposes, therefore, we may regard the eye as having a single nodal-point. By calculation it is shown that this point lies very near the posterior surface of the crystalline lens, about 7 millimetres behind the cornea.

Inversion of the Image.—It is apparent from Fig. 11 that the retinal image is always inverted with respect to the object of vision. The mind, however, takes no cognizance of this inversion, since it possesses the power of external projection, so that we see, not the image, but the object in its true position. This power has doubtless been derived through association with the sense of touch. We have learned that a stimulus conveyed to the brain from the upper part of the retina proceeds from an object situated below the eye, and *vice versa,* and that a stimulus on the temporal side of the retina must proceed from an object on the nasal side of the eye, and *vice versa.*

FIG. 11.



Aberration.—We have assumed that all the rays proceeding from a point are united in its conjugate focus after refraction at a spherical surface. This is true only of small pencils of light whose peripheral rays are not much removed from the optic axis. It is found by calculation and experiment that the peripheral rays are relatively too strongly refracted, and consequently meet the axis sooner than the more central rays. This constitutes *spherical aberration;* it is apparent that the effect of aberration is to mar the sharpness of images. The refracting surfaces of the eye, though not strictly spherical, may practically be so regarded, at least at their central portions, which alone are normally concerned in refraction, since the iris prevents the more peripheral rays from reaching the retina.

In certain cases, especially in conical cornea, the peripheral refraction is relatively insufficient, the focal distance for peripheral rays being longer than that for the more central rays. This is called *negative aberration.*

There is still another kind of aberration, which is due to the fact that the various colors are not equally refracted, violet being the color of greatest and red that of least refraction. This is *chromatic aberration.*

Aberration does not ordinarily interfere with vision, partly because the iris cuts off the more peripheral rays, and partly because the retina is so constructed that a well-defined visual impression is conveyed to the brain only when the stimulus falls upon the macula lutea, which is so situated as to receive images only from points lying near the optic axis of the eye. While the peripheral portion of the retina is very useful in extending the field of vision, it is only the macula and more especially the fovea that is directly concerned in distinct vision.

Angles Alpha and Gamma.—In addition to aberration, the eye is subject to certain other imperfections. The various surfaces are not perfectly centred on a single line or axis, as in a perfectly constructed optical apparatus, and the fovea does not lie exactly on the line which passes through the middle of the cornea and the centre of rotation of the eye,—the line which most nearly represents a common optic axis of the various surfaces. Since the image must fall upon the fovea in direct vision, it is apparent that the line connecting the point of most acute vision with its image on the fovea and passing through the nodal-point of the eye must be obliquely inclined to the axis; that is, the axis of direct vision is a secondary axis. This line is called the *line of vision.* The angle which the line of vision makes with the optic axis at the nodal-point is called *gamma.* The angular distance of the fovea from the optic axis usually ranges from 2° to 4°, chiefly in the horizontal meridian, the fovea being usually displaced temporally as regards the optic axis. When this is the case it is apparent that the visual line must intersect the cornea on the nasal side of the optic axis. Furthermore, it is found by keratometric examination that the cornea is approximately a segment of the small end of an ellipsoid, and that the corneal summit, or point where the major axis of the ellipsoid cuts the cornea, usually lies slightly on the temporal side of the optic axis. The angle which the major axis of the cornea makes with the visual line is called *alpha*.

Accommodation.—We have learned that as an object approaches a refracting surface the conjugate image on the opposite side of the surface recedes from the surface. Hence if the eye is of such length that light from a distant object is focused on the retina, it is evident that light from a near object must impinge upon the retina before the rays have been united in a focus, unless the refractive power of the eye can be suitably increased. The eye does possess the property (except in old age) of increasing its refractive power. It accomplishes this by an increase in the convexity of the crystalline lens, chiefly of the anterior surface. This power which the eye possesses of varying its refractive power to adapt itself for vision at different distances is called *accommodation*. So long as the object of vision is distant more than about 6 metres (20 feet) no accommodation is required; that is, for all distances not less than this limit the posterior conjugate focus coincides practically with the principal focus; or all distances not less than 6 metres may be regarded as infinite in our study of the refraction of the eye.

Accommodation is effected by contraction of the ciliary muscle as the result of reflex stimulation received by the accommodation centre from its connection with the optic nerve, probably through the corpora quadrigemina. The accommodation centre constitutes the anterior portion of the nucleus of the third nerve. From this centre an efferent impulse is transmitted through the ciliary ganglion to the ciliary muscle, and at the same time an impulse is sent to the sphincter pupillæ, so that contraction of the pupil takes place simultaneously with accommodation.

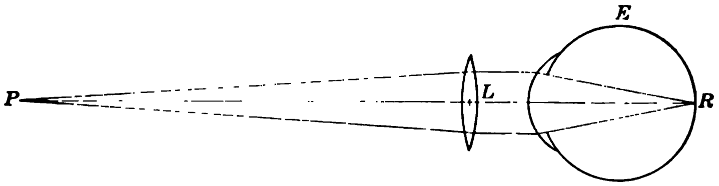
There are two theories as to the manner in which contraction of the ciliary muscle produces an increase of convexity of the crystalline lens. According to Helmholtz's theory, which until recently was universally accepted, contraction of this muscle draws forward the anterior part of the chorioid and reduces the circumference of the ciliary muscle with a resultant relaxation of the suspensory ligament of the lens. The latter then by the inherent property of soft matter inclosed in a capsule and subjected to a uniform outside pressure assumes a more spheroidal shape with a marked increase of convexity of its anterior surface.

Tscherning, on the other hand, has endeavored to prove that contraction of the ciliary muscle causes (through the meridional fibres) an increased tension upon the suspensory ligament, and that because of the resistance of the nucleus of the lens there is a resulting increase of curvature at the axial part of the lens with a diminution of curvature at the periphery. Since this theory requires the assumption of a firm nucleus, it would seem untenable, for we know that no such resistant nucleus exists in childhood,—the period of greatest accommodative activity.

Range of Accommodation.—The nearest point for which an eye can adapt itself is called the *near-point* of the eye, and the distance through which distinct vision is possible is called the *range* or *amplitude of accommodation.* In the normal eye this embraces the whole region from infinity to the near-point. The farthest point of distinct vision is called the *far-point* of the eye.

Measurement of Accommodation.—Accommodation may be measured by the convex lens which, when placed in front of and as near the eye as possible, enables light from a point to be focused on the retina without the exercise of accommodation. This lens is the equivalent of the accommodation which must be exercised to enable the light from the same point to be focused on the retina without the lens. Thus if *E*, Fig. 12, represents an eye adapted for an infinite distance, parallel rays will be focused on its retina, and in order that rays from a near-point, *P*, may be focused on the retina, the eye must exercise accommodation, or else a convex lens, *L*, whose focal length is *LP*, must be placed before the eye. Since *P* is the anterior principal focus of the lens, rays from *P* will be parallel after passing through the lens, and will, consequently, be focused at *R* on the retina of the eye, which is adapted for parallel rays. Hence the lens whose focal length is *LP* is the equivalent of the accommodation which must be exercised by a normal eye in order that rays from *P* may be focused on the retina. If *P* is the nearest point

FIG. 12.



for which the eye can accommodate, the lens whose focal length is *LP* is the lens-equivalent of the accommodative power of the eye.[2]

Presbyopia.—As the lens hardens with increase of age there is a gradual diminution of accommodative power. The following table (Donders) gives the average accommodative power at different ages:

Age	10	15	20	25	30	35	40	45	50	55	60	65	70
Diopters	14	12	10	8.5	7	5.5	4.5	3.5	2.5	1.75	1	0.75	0.25

It is shown in this table that at 45 years of age the amplitude is 3.5 D., which represents in a normal or emmetropic eye (adapted for an infinite distance without accommodation) the power of focusing on the retina rays from all objects at or beyond a distance of $\frac{1}{3.5}$ metre (9 inches). The image of an object nearer than this limit will be blurred. In order that a very small object, such as fine printed matter, may be clearly seen it must be held near the eye, so as to form on the retina an image large enough to be deciphered, and when the focusing power does not afford a clear image at a distance of 22 centimetres, or 9 inches, the reading of very small print becomes difficult. Furthermore, it is not possible to use continuously all the accommodative power. It is found that one can use for a prolonged period only about two-thirds of the total amplitude; when he has not one-third in reserve great fatigue (asthenopia) quickly ensues. Hence, in order that any one may use his eyes continuously for work at a distance of one-third of a metre (33 centimetres, or 13 inches) he must have a total accommodative power of 4.5 D.—3 D. for use and 1.5 D. in reserve. When the amplitude falls below this amount the condition is called *presbyopia* (old sight). Presbyopia is, therefore, that condition in which, from physiological sclerosis of the lens, the accommodative power is less than 4.5 D. It is shown in the table that this condition makes its onset about the fortieth year. Usually, however, artificial assistance is not required before the forty-fifth year. At this age a convex spherical lens of 1 D. generally affords the necessary assistance. The probable strength of lens required to overcome the presbyopia at different ages is given in the following table:

Age	45	50	55	60	65	70	75
Diopters	1	2	2.75	3.25	3.5	3.5	3.5

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[2] The power of a lens is inversely proportional to its *focal length*, which is the distance from the lens of the focus for parallel rays; the shorter the focal length, the greater is the refractive power of the lens. The unit of lens measure in ophthalmology is the Diopter, which expresses the power of a lens whose focal length is one metre. Similarly, 2 D. is the power of a lens whose focal length is ½ metre; 3 D. is the power of a lens whose focal length is ⅓ metre, etc.

Obviously this table serves only as a guide. It is not to be strictly followed in the selection of working-glasses. The determination of the latter depends not only upon the distance at which work is most conveniently done, but also upon the refractive condition of the eye; that is, the strength of the working-lens depends upon the position of the far-point, or point of adaptation in relaxation of the accommodation. In hyperopia, as will be subsequently shown, a convex lens is required to adapt the eye for distant vision without accommodation. It is evident, therefore, that in the selection of a glass for near work the correction of the hyperopia must be added to that of the presbyopia. In myopia, on the other hand, a concave lens is required to adapt the eye for distant vision, and in this case the difference between the distance-glass and the presbyopic correction represents the strength of lens required for near work.

ANOMALIES OF REFRACTION.

In emmetropia, the typically normal optical condition, parallel rays are focused on the retina without accommodation; that is, the eye is adapted for distant vision, and by the exercise of accommodation it is adapted for near objects. This is the most favorable condition for the varying requirements of civilized life, but since it necessitates an exact correspondence between the refractive power of the eye and the length of the eyeball, emmetropia, as determined by refined methods of measurement, is the exception rather than the rule. When the posterior principal focus does not fall upon the retina in relaxation of the accommodation the eye is said to be *ametropic* or to have an *error* or *anomaly* of *refraction.*

There are three kinds of ametropia: *hyperopia* or *hypermetropia,* in which the posterior principal focus lies behind the retina; *myopia,* in which the focus lies in front of the retina; and *astigmia,*[3] in which, owing to asymmetry or irregularity of one or more of the refracting surfaces or to heterogeneity of structure, the rays are not brought to a focus at any point.

Hyperopia.—Hyperopia may be due to any one of three causes: (1) deficient curvature of the cornea or lens (*curvature hyperopia*);

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[3] The writer has pledged his support to the endeavor now being made by Dr. Swan Burnett and others to replace the commonly employed word *astigmatism* by the more euphonious and correct word here used. The impropriety of the former word has long been known. The argument in favor of making a change at this time is ably presented by Dr. Burnett in the American Journal of Ophthalmology, December, 1903.

(2) abnormal refractive index, such that the total refractive power of the eye is too low (*index hyperopia*—very rare); and (3) abnormally short antero-posterior diameter of the eyeball (*axial hyperopia*). By means of many measurements which have been made it has been proved that axial hyperopia is by far the most common variety.

In hyperopia the refractive power of the eye in relaxation of the accommodation is insufficient to focus rays from any point, distant or near, on the retina (Fig. 13). The deficiency of refraction may be overcome by means of a convex spherical lens placed in front of the eye. This is illustrated in Fig. 14, in which parallel rays from a distant point, *O*, would be focused by the hyperopic eye, *E*, at some point behind the retina, but with the aid of the lens, *L*, the rays are focused on the retina, *R*. It is apparent from this diagram that *LP* is the focal length of the lens, *L*, which corrects the hyperopia, and that this focal length increases as the lens is moved farther from the eye, since *P* is a fixed point, conjugate to the retina. That lens which, when placed as near as possible to the eye (the position in which spectacles are worn), enables rays from a distant point to be focused on the retina is taken as the measure of the hyperopia. Thus, if *LP* is 1 metre there is hyperopia of 1 D.; if *LP* is ½ metre there is hyperopia of 2 D., etc.

FIG. 13.

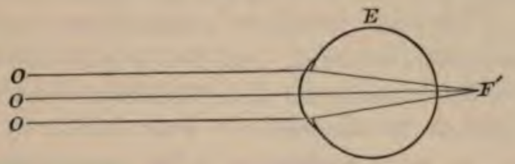
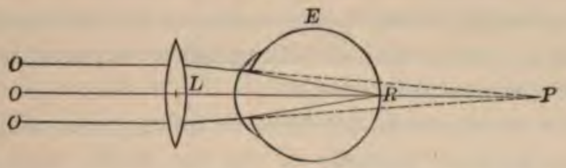


FIG. 14.



In the study of accommodation we learned that the eye is able to increase its refractive power by increasing the convexity of the crystalline lens. In emmetropia this power is called into play only in near vision, but in hyperopia, when uncorrected by a lens, accommodation must be exercised to render distant vision distinct, and in near vision the same degree of accommodation which would be required of a normal eye must be used in addition to that required for distant vision. It is apparent, therefore, that the unaided hyperopic eye has an abnormal task imposed upon it, both in distant and in near vision. In childhood when the accommodative power is at its maximum a moderate degree of hyperopia is very often not attended by any disturbance, the accommodation being ample for near as well as for distant vision, but when with advancing age the accommodative power diminishes, the tax imposed upon the ciliary muscle gives rise to various symptoms of nerve-exhaustion (asthenopia).

So easy is accommodation in childhood that it is usually not possible for a hyperope at this period of life to relax all of his accommodation when using his eyes. Hence a portion of the hyperopia will be masked by accommodative action, and it is only by the employment of a cycloplegic or by examination in a dark room that the full extent of the hyperopia can be ascertained. That portion of the hyperopia which is masked by accommodation is called *latent* hyperopia. Hyperopia which exists in excess of what is latent is called *manifest;* that is, manifest hyperopia is measured by the strongest convex lens which does not impair distant vision. The sum of the latent and manifest hyperopia constitutes the *total* hyperopia. In childhood hyperopia of a low or moderate degree is very frequently all latent, but sooner or later, the age varying with the degree of hyperopia and with the physical condition, a portion becomes manifest, and this portion continually increases at the expense of the latent hyperopia.

There is, further, a division of manifest hyperopia into that which can be overcome by accommodative action to render distant vision distinct (*facultative hyperopia*), and that which cannot be so overcome (*absolute hyperopia*). In old age the total hyperopia is absolute, so that distant vision can be made distinct only by the help of a convex lens. In near work an absolute hyperope would require the appropriate presbyopic correction added to the correction of the hyperopia. Thus in hyperopia of 2 D., a lens of 5 D. would be required for reading at a distance of $\frac{1}{3}$ metre (13 inches).

Hyperopia is the normal condition in infancy, but with the increase of the antero-posterior diameter of the eye which takes place in the process of growth, hyperopia is succeeded by emmetropia or myopia prior to adult life in about one-half of all persons in civilized countries. Among savages hyperopia is the rule at all ages.

It is apparent, therefore, that hyperopia (except in the higher grades) is, like presbyopia, a physiological condition; yet, owing to the great preponderance of near-work which results from the requirements of civilization, it is in most cases necessary to correct all or a portion of the defect before the hyperope has advanced far in adult life. In fact, even a moderate hyperopia may require correction in childhood to relieve the strain which is imposed by the unnatural near-work incident to school life.

In the treatment of hyperopia a number of questions arise, the most important of which may be summarized as follows: (1) As to when it is advisable to correct the defect; (2) as to the portion of the defect to be corrected; and (3) as to whether the glasses should be worn constantly or for near-work only.

As to the first question, hyperopia does not require correction unless it gives rise to some physical disturbance, as inability to see clearly, asthenopia, headache, or convergent strabismus; but since few persons consult a physician except when suffering from physical disability, it is apparent that almost all cases of hyperopia which are brought to the notice of the physician require correction.

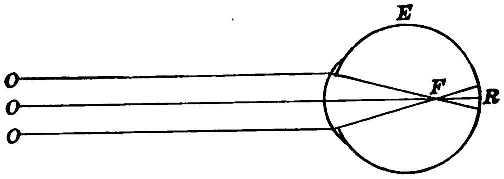
As to the portion of error to be corrected, it is not possible to give a fixed rule. Usually in childhood the lens should not exceed one-half or two-thirds of the total error as determined with the eye under the influence of a cycloplegic. The chief exception to this rule is when the hyperopia is complicated with convergent strabismus, when all or nearly all the error should be corrected. In adults correction of the greater part of the hyperopia will usually be tolerated.

Still less is it possible to answer the third question by a fixed rule. In general, children who suffer from asthenopia resulting from a low grade of hyperopia should wear correcting glasses during school and study hours only; but when the error reaches 2.5 D. or 3 D. it is usually found that the ciliary muscle will not adapt itself to the use of glasses unless the latter are worn constantly. Similarly in strabismus relief is not to be expected unless the glasses are worn constantly. In adult hyperopes who suffer from nerve-strain, as the result of prolonged near-work, the same general rule is applicable; that is, the glasses may be worn for near-work only, if the error is slight, and especially if it is latent, but for higher grades of the defect the glasses must be worn constantly.

Myopia.—Myopia is divisible into *curvature myopia*, *index myopia*, and *axial myopia*. Curvature myopia results from keratoconus or conical cornea, or from increased convexity of the lens in partial dislocation (when released from traction by the suspensory ligament). Myopia also occurs not infrequently as a premonitory sign of senile cataract, and in diabetes. This may be due either to increase of convexity of the lens from swelling or from increase of refractive index of the nuclear portion of the lens. With these exceptions myopia is almost always due to excessive length of the antero-posterior diameter of the eyeball.

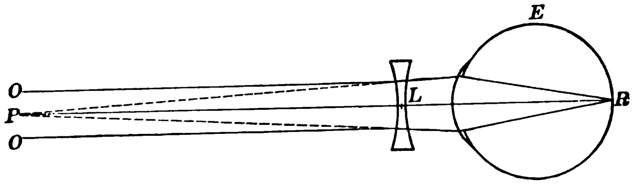
In myopia, whatever its cause, the refractive power of the eye is too great in proportion to the axial length, and consequently rays from a distant object (parallel rays) will be united in a focus before they reach the retina, and the image of a distant object as formed on the retina will be blurred (Fig. 15). The predominant symptom of myopia is, therefore, the inability to see distant objects clearly; and since only near objects are clearly distinguished, the affection is popularly called *near-sightedness.*

FIG. 15.



An object situated at *P* (Fig. 16), which is conjugate to the retina, and which is the far-point of the eye, can be clearly seen without any accommodation, and an object nearer the eye than this point can be seen with less accommodation than would be required by a normal eye, whose far-point is at infinity. This is not, however, as it might appear, always an advantage in near-work, for it not infrequently leads to severe strain on the convergence centres, the nervous mechanism being so constituted that exercise of accommodation is required as an incentive for the proper convergence of the visual lines of the two eyes, so that the image may in each case fall upon the fovea. Furthermore, when the myopia exceeds 3.5 D. or 4 D., the farthest point of distinct vision is so near the eyes that the amount of convergence required is too great to be comfortably maintained; in uncorrected high myopia, vision is performed with one eye, while the other assumes a position of relative or absolute divergence (divergent strabismus).

FIG. 16.



Axial myopia, which, as previously stated, is the typical form of myopia, is divisible into two classes. The first class embraces those cases in which the eyes are hyperopic prior to the beginning of school life. In the natural course of growth there occurs an enlargement of the eyes with consequent diminution of hyperopia, and, normally, hyperopia passes into emmetropia before adult life. But in a certain proportion of cases the increase in the axial length is not arrested when emmetropia is reached, and a condition of myopia results. This is probably caused by stretching of the sclera by muscular pressure and traction in the convergence required in school work. This kind of myopia does not reach a very high degree and it ceases to advance, after the sclera has acquired its normal resisting power, in adult life. It is, therefore, called *benign* or *school myopia.*

The second class of axial myopia is due to defective development of the sclera; the myopic condition is present at an early age—prior to the beginning of school life. It may reach a very high degree (*posterior staphyloma*), and together with the distention of the sclera there may occur atrophy of the chorioid, vitreous opacities, hemorrhages, or detachment of the retina, with partial or total loss of vision. This kind of myopia is, therefore, appropriately called *malignant myopia.*

The malignant variety is fortunately much less common than the benign. While the latter is confined chiefly to the student classes, the former occurs in a relatively greater proportion among the poorly developed lower classes. The proportion of myopes varies in different countries. In America statistics have shown a proportion of about 25 per cent. among college students, while in Germany the proportion among the same class of persons reaches 50 per cent.

As in hyperopia, the deficiency of refraction is overcome by the convergent action of a convex lens, so in myopia the excess of refraction may be overcome by the divergent action of a concave lens. Thus if L (Fig. 16) is a concave lens, the parallel rays which meet it are rendered divergent so that they appear, after passing through the lens, to proceed from the point P. Hence if P is conjugate to the retina of the myopic eye E, it is apparent that parallel rays which are made to pass through the lens will enter the eye E as if coming from P, and will consequently be focused on the retina. An appropriate concave lens will, therefore, make distant objects appear distinct to the myopic eye. The eye may then see near objects with the aid of accommodation, or the lens may be removed in near vision.

It is apparent from Fig. 16 that parallel incident rays will never be united in a focus as the result of refraction by a concave lens, but P

is the point on the axis through which, after refraction, the rays appear to pass. The point P is, therefore, the *virtual focus* (for parallel rays) of the concave lens, in contradistinction to the real focus of the convex lens. The focal length of the lens L is LP; it is expressed in diopters, as was explained for the convex lens; but since P lies on the left of the lens, instead of on the right as in the convex lens, the focal length is regarded as negative, and the refractive value is expressed by the minus (−) sign, which is used before the dioptric number.

The degree of myopia is measured by the focal length or dioptric power of the lens, which, when placed as near as possible to the eye, focuses parallel rays on the retina. Thus if LP is 1 metre, the eye has 1 D. of myopia; if LP is $\frac{1}{3}$ metre, the eye has 3 D. of myopia, etc.

In the adaptation of concave lenses to the correction of myopia the general rules differ somewhat from those given for the correction of hyperopia. Myopia occurring in young children should be corrected at as early an age as is compatible with the wearing of spectacles, not only for the beneficial effect in retarding the advance of the myopia (probably by relieving the eyes of the necessity of excessive convergence), but also for the purpose of placing the child under favorable circumstances in cultivating his powers of observation. All, or all but a small fraction, of the myopia should be corrected, and the glasses should be worn both for distance and near-work. In the case of adults, however, when the myopia has not been corrected in childhood, the ciliary muscle is poorly developed, and the full amplitude of accommodation is not attained. In such cases it often happens that the glasses which are suitable for distance will not be tolerated for near-work; for the latter a weaker concave lens, or no lens, or a weak convex lens may be required, according to the degree of myopia and the age of the individual.

In the highest grades of myopia (exceeding 15 D.) the myopia may be overcome or greatly reduced by extraction of the crystalline lens.

Of no less importance than the correction of myopia is its prophylaxis by the inculcation of proper hygienic principles, such as the avoidance of close application of the eyes at a very early age, the necessity of good illumination of school rooms, and of large and clear print for young children.

Astigmia.—Astigmia is that condition in which rays proceedin
from a point are not united in a focus at another point aft
There are two kinds of astigmia of the eye: (1) That
asymmetrical, but regular, curvature or oblique posi

or lens;[4] and (2) that which is due to unevenness or irregularity of one or more of the surfaces, or to heterogeneity of composition of the crystalline lens. The first kind constitutes *regular* and the second *irregular* astigmia.

Astigmia exists, to a slight extent, as a physiological imperfection in all eyes. The curvature of the cornea is, as a rule, somewhat greater in the vertical than in the horizontal meridian. On the other hand, the crystalline lens presents the opposite condition, for, on account of a slightly oblique position, its meridian of greatest refraction is usually horizontal. These two defects of curvature tend to neutralize each other, but as a rule the vertical refraction predominates, thus giving rise to a slight degree of regular astigmia. Furthermore, the normal crystalline lens is not free from heterogeneity of structure, for, being composed of a number of segments, and each segment of a number of fibres, its refraction is considerably marred by irregularity, which is the chief cause of the normal irregular astigmia.

Regular astigmia, exceeding the amount which may be regarded as normal or physiological, may be due to excess of vertical over the horizontal curvature (*astigmia with the rule*); or to excess of horizontal curvature (*astigmia against the rule*); or to asymmetry in which the meridians of greatest and least curvature (these being at right angles to each other) are neither vertical nor horizontal (*oblique astigmia*). As may be inferred from the normal asymmetry of the cornea and lens, astigmia with the rule is more commonly the result of corneal asymmetry (*corneal astigmia*), while astigmia against the rule is more commonly due to asymmetry of the lens (*lenticular astigmia*); but this is by no means always the case, for quite often the meridian of greatest corneal curvature is horizontal, thus giving rise to astigmia against the rule. This is notably so after cataract operations, since the section of the cornea leaves a resulting diminished curvature in the vertical direction without materially altering the curvature of the horizontal meridian.

Regular astigmia, since it results from a regular asymmetry of curvature, in which the meridians of greatest and least curvature (*the principal meridians*) are at right angles to each other, may be overcome by a cylindrical lens, which has the property of refracting rays in the meridian of its curvature without altering their direction in the plane of the axis of the cylinder. Hence if the refraction of an eye is such that rays proceeding from a distant point are, in the vertical meridian, focused on the retina, while in the horizontal meridian of less curvature the rays are intercepted by the retina before they have reached their focus, a suitable convex cylindrical lens, having its refracting meridian horizontal (its axis vertical), will so add to the refraction in the horizontal meridian that the rays will be focused on the retina in this as well as in the vertical meridian; in other words, the rays will be focused in a point on the retina and the astigmia will be overcome. When, as in this case, the eye is emmetropic in one meridian and hyperopic in the other, the condition is called *simple hyperopic astigmia.*

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[4] The astigmia which results from obliquity of the cornea or lens with reference to the optic axis is usually classed as regular since the defect of vision resulting therefrom can be improved by a cylindrical lens; but from a mathematical point of view the condition differs somewhat from that which results from asymmetrical curvature.

If with the same asymmetry of curvature the eyeball is slightly larger than in the former case, so that the horizontal focus falls on the retina while the vertical focus is in front of the retina, the condition is *simple myopic astigmia.* In this case rays will be focused in a point on the retina with the aid of a suitable concave cylindrical lens, axis horizontal, since this will throw the vertical focus back to the retina without affecting the rays in the emmetropic horizontal meridian.

If both foci are behind the retina, one farther than the other (*compound hyperopic astigmia*), a convex spherical lens must be added to the appropriate cylindrical lens in order to bring the focus on the retina. If, on the other hand, both foci are in front of the retina (*compound myopic astigmia*) a concave spherical lens must be added to the appropriate cylinder. It may happen evidently that the retina is situated between the two foci, one of these being in front of the retina and the other behind it (*mixed astigmia*). In this case a convex cylinder, such as will equalize the refraction in the two principal meridians and thus bring the rays to a focus in front of the retina, may be combined with a concave spherical lens of sufficient strength to throw the focus back upon the retina; or a concave cylinder, such as will equalize the refraction [illegible] focus behind the retina, may be combined with a [illegible] bring the focus forward upon the retina.[5]

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[5] [illegible] for the correction of compound and mixed astigmia [illegible] ground upon one side of the glass and the cylindrical upon [illegible] compound astigmia both surfaces should be convex [illegible] the condition is that of hyperopic or myopic astigmia, [illegible] astigmia one surface must be convex and the other [illegible] ylinder is the common form of lens used in the correction of [illegible] it is possible [illegible] stitute for this form of glass a crossed- [illegible] ylinder on [illegible] the glass, the two axes being at right [illegible] a toric lens, in wh[illegible] asymmetrical curvature is ground upon [illegible] of the glass.

The degree of astigmia is measured by the dioptric power (in the refracting meridian) of the cylindrical lens which equalizes the refraction. The degree may vary from .12 D. (the weakest lens commonly made) to 15 D. or more. Almost all eyes have as much asymmetry as is indicated by the former limit, but the higher degrees are fortunately uncommon. In fact, astigmia of more than 5 D. or 6 D. is not often encountered. In the lower grades of asymmetry the effect upon vision is very slight, and in many such cases no disturbance of any kind results; on the other hand, in persons who are engaged in close application of the eyes astigmia of .25 D. or even .12 D. may produce asthenopia, which is relieved only by the use of correcting lenses.

The treatment of regular astigmia consists in the adaptation (as will subsequently be explained) of the appropriate cylindrical lens. In the lower grades the lens-correction may be worn only in close application of the eyes, but in the higher grades, preferably in all reaching 1 D., the correction should be worn constantly.

Irregular astigmia exceeding the physiological amount is most commonly due to scar formation in the cornea or to irregular swelling of the crystalline lens in the premonitory stage of cataract. This kind of astigmia is incapable of correction.

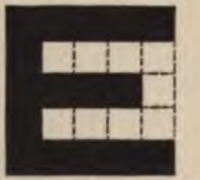
Anisometropia.—Since each individual is normally endowed with a pair of eyes, which work together in the function of vision, the relative refractive condition of the two eyes is a matter of great importance. The ideal condition is evidently that in which the refraction is alike in the two eyes; this is called *isometropia.* But more commonly an absolute correspondence of the two eyes is not found; one eye may be emmetropic and the other hyperopic, myopic, or astigmic; or both eyes may have the same defect, the degree being higher in one eye than in the other; or one eye may have hyperopia and the other myopia. Any such deviation from isometropia is called *anisometropia,* and the special condition in which one eye is hyperopic and the other myopic is usually called *antimetropia.* While it is, in most cases, possible to detect a slight difference between the two eyes, it is only when the disparity is marked that anisometropia should be regarded as an anomaly, capable of giving rise to asthenopia or other visual disturbance.

In the treatment of anisometropia the ophthalmologist endeavors to equalize the condition, as far as practicable, in the two eyes by giving to each eye its appropriate correcting lens. In childhood quite a high disparity may thus be overcome, but in those cases which have not been corrected prior to adult life the correction of the defect will, in the more marked cases, rarely be tolerated. In some of these cases vision is performed with both eyes, the clear image formed in one eye being fused with the blurred image formed in the other, but in very many cases vision is uniocular, the better eye being used, while the poorer eye falls into a state of relative deviation or squint. On this account it is of the utmost importance that anisometropia should be corrected in early childhood.

Determination of Visual Acuteness and of the Refractive Condition of an Eye.

The visual power is measured by the least interval which permits the eye to distinguish as such two bright or white points on a black ground. In the normal eye this minimum interval subtends an angle of about one minute at the nodal-point. This angle is called the *minimum visual angle.* For practical vision-testing it is more convenient to use a series of test-letters, so constructed that when they are placed at the proper distance, the breadth of each stroke of the letter subtends the minimum visual angle at the nodal-point of the eye undergoing examination, while each dimension of the whole letter subtends an angle of five minutes (Fig. 17). For determining visual acuteness cards are furnished having printed on them letters of various sizes, the distance at which each letter subtends the five-minute angle being noted on the card. Thus, if at a distance of six metres, a person can distinguish those letters which, as marked on the card, subtend the five-minute angle at this distance, the visual acuteness is normal ($V = \frac{6}{6}$ or $V = 1$); but if at this distance the smallest distinguishable letters are those which subtend the five-minute angle at twelve metres, the visual power is only $\frac{6}{12}$ of the normal acuteness ($V = \frac{6}{12}$). The visual acuteness of any eye is therefore measured by a fraction whose numerator is the distance at which the test is conducted and whose denominator is the distance at which the smallest distinguishable letters subtend the five-minute angle.

Fig. 17.



It not infrequently happens, especially in young persons, that the visual power exceeds the standard which has been accepted as the average vision of normal eyes. We thus have the expressions $V = \frac{6}{5}$, or $V = \frac{6}{4}$.

The minimum visual angle is the basis of the most valuable method of determining the refractive condition of the eye. The refraction of the eye indicates the relation between the position of the retina and posterior focus for parallel rays, *when the accommodation is relaxed.*[6] Hence in determining the refraction the test-letters must be at such a distance that the rays proceeding therefrom may be regarded as parallel; that is, the letters must be five or preferably six metres (20 feet) from the eye undergoing examination. With the letters at this distance normal vision is possible only in emmetropia or in hyperopia with use of accommodation, since full visual acuteness necessitates an accurate focus of the image on the retina. To decide between these two conditions we place a weak convex spherical lens before the eye under examination (the other eye being excluded from vision by an opaque disk); if this lens does not render vision worse (hazy) accommodation has been replaced by the convergent action of the lens. By trying successively stronger lenses we find the strongest lens which does not impair vision. This measures the manifest hyperopia. In old persons it also measures the total hyperopia, but in children and young adults a certain portion of the hyperopia is, as we have learned, latent. In fact the whole of the hyperopia may be latent, so that the weakest convex lens will make vision worse. When latent hyperopia cannot be otherwise excluded a cycloplegic must be instilled into the conjunctival sac, and the examination made while the eye is under the influence of the drug. If under this condition vision remains normal, the eye is emmetropic; but if the vision, which before was normal, falls below normal during cycloplegia, the convex spherical lens which affords maximum vision measures the total hyperopia.

Homatropin-hydrobromate is the most suitable cycloplegic for general use; one drop of a one-and-one-half per cent. solution instilled at ten-minute intervals until six applications have been made will, except in rare instances, produce complete paralysis of the accommodation in one hour after the last instillation. The effect of the drug passes off in about twenty-four hours. In young children and in other persons in whom a prolonged enforced rest of the accommodation is desirable atropin may be used. To insure complete paralysis, in excessive accommodative activity, one drop of a one per cent. solution of atropin-sulphate may be instilled three times a day for several days, though usually complete paralysis occurs in one and one-half hours after the first instillation. The full accommodative activity is not restored until about two weeks after the last instillation.

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[6] This is the *static* refraction, in contradistinction to the *dynamic* refraction, which refers to the accommodative power.

The use of a cycloplegic is contraindicated when there is any tendency to glaucoma; but as this affection seldom occurs in young persons, who alone require a cycloplegic for the determination of refraction, there is ordinarily no danger in the use of a cycloplegic for this purpose.

If vision is less than normal at the first examination (without cycloplegia), the defect of vision is due, if to any error of refraction, either to hyperopia without sufficient accommodation to overcome it, or to myopia, or to astigmia. The first of these conditions is determined by the improvement effected by placing a convex spherical lens before the eye. On the other hand, the presence of myopia is revealed by the fact that vision is rendered worse by a convex lens and improved by a concave lens. But while in hyperopia the strongest acceptable lens measures the ametropia, in myopia the weakest lens which affords maximum vision must be selected, for if a stronger lens is taken, normal vision may still be possible with the aid of accommodation. In fact, so active is accommodation in young subjects that overaction of the ciliary muscle (*spasm of accommodation*) sometimes gives rise to an apparent myopia, whereas the true condition is that of emmetropia or hyperopia. When there is any reason for suspecting an overaction of the ciliary muscle the refractive condition should be determined while the eye is under the influence of a cycloplegic. In fact it is better in most cases to use a cycloplegic for the determination of refraction in persons under thirty years of age, since by this means only can the static refraction be determined with absolute certainty.

When neither a convex nor a concave spherical lens brings the vision up to the normal standard, we suspect the presence of astigmia. If this is regular it may be corrected by a cylindrical lens. If a spherical lens has been found which improves vision, this should remain before the eye in the trial frame in conducting the examination for the determination of the existence of astigmia. In the latter procedure we place a weak convex cylindrical lens before the eye; and, since more commonly the eye has its greatest curvature in the vertical meridian, we place the axis of the lens in this meridian. If in this position it does not improve vision, we rotate the lens to ascertain whether in any other position it effects an improvement.[7] If we thus succeed in finding a position in which the lens improves vision, we try stronger lenses until we find that lens which gives normal vision. If the weak convex cylinder is not accepted in any meridian, we next try a concave cylinder. In this case we first place the axis in the horizontal meridian, since the probability is that the vertical curvature is excessive. We then proceed as with convex cylinders until we find the position and the lens which give best vision. If we have difficulty in determining the directions of the principal meridians by the foregoing method, we may make use of the *clock-face* chart (Fig. 18), which consists of a series of radiating lines, numbered in the same manner as a clock face. If (after correction of any existing hyperopia or myopia) one group of these lines appears very distinct while the other lines, and especially the group at right angles to the group of greatest distinctness, are blurred, the eye is astigmic, and the lines of greatest distinctness lie in the meridian which requires correction.[8] Hence the axis of the cylindrical lens should be placed in the direction of the most blurred lines. If a convex lens having its axis in this direction does not improve vision, we try a concave lens with its axis in the same direction. The lens which equalizes all the lines and at the same time affords normal vision is the correction of the astigmia.

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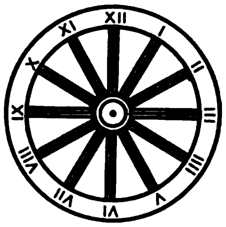
[7] The position of a cylindrical lens is registered in accordance with the direction of its axis, as indicated by the angular marking on the trial frame.

FIG. 18.



While simple refractive errors in eyes having normal visual acuteness may be easily measured by the method of examination just described, those cases in which hyperopia or myopia is complicated by astigmia, and especially in eyes having imperfect visual acuteness, much time may be saved and greater accuracy secured by the aid of *objective*

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[8] If, for instance, the eye is emmetropic in the horizontal meridian, but not so in the vertical meridian, a distant point will have its horizontal focus on the retina, while in the vertical meridian the rays will be diffused on the retina, that is, the image of a distant point will be a vertical line; therefore since every point of a vertical line will have a corresponding vertical line as its image, the image will be a sharply defined line. But if the line is horizontal instead of vertical, that is, if the line lies in the emmetropic meridian, every point of the horizontal line will have a short vertical diffusion line as its image, and the image of the whole line will be diffused and blurred.

methods of examination. The most valuable of these methods are *ophthalmoscopy*, *skiascopy*, and *keratometry*.

The refractive condition may be determined, with a degree of accuracy varying with the experience and skill of the examiner, by *direct ophthalmoscopy*. If the examiner is emmetropic (or has his ametropia corrected by a lens) the hyperopia of the examined eye is measured by the strongest convex ophthalmoscopic lens with which the small retinal vessels can be distinctly seen; while myopia is measured by the weakest concave lens with which these vessels are distinctly seen. Astigmia is measured by the difference between the lens which corrects the ametropia in one principal meridian (as determined by distinctness of the retinal vessels at right angles to this meridian) and the lens which corrects the ametropia in the other principal meridian. The accurate estimation of astigmia in this way is very difficult; in fact, it is not usually attempted, since the astigmia can be much more easily determined by skiascopy.

Skiascopy consists in the estimation of the refractive condition from the movement of the shadow which passes across the pupil of the examined eye when the illuminating mirror is rotated. The movement of this shadow varies with the position of the observer (who looks through the sight hole of the mirror), with reference to the far-point of the examined eye. If the examiner is at this point there is no perceptible movement of the shadow, but, as the mirror is shifted, the whole pupil is simultaneously enshrouded in darkness. If the examiner is nearer the eye than the far-point, he sees a shadow move across the pupil in the direction in which he rotates the mirror, the rate of movement diminishing as he departs from the far-point. If, on the other hand, he is farther from the eye than the far-point, he sees the shadow move in the opposite direction to the mirror rotation.[9] The explanation of this change of movement is, found in the fact, that the rays proceeding from any point of the retina meet at the far-point of the eye and have their relative position reversed in passing this point. On this account the far-point is called the *point of reversal* of the eye. We know that in emmetropia the far-point (the anterior conjugate to the retina) is at infinity, and in hyperopia it is negative or behind the retina; hence it is only in myopia that the examiner can place himself at the point of reversal. If, for instance, he finds that there is no perceptible shadow at a distance of one metre, while the shadow moves opposite to the mirror when he moves to a greater distance, and in the same direction as the mirror when he moves nearer the eye than the one-metre limit, he knows that the eye has 1 D. of myopia, since the degree of myopia is measured by the distance of the far-point from the eye (Fig. 16). But if as the examiner moves away from the eye the shadow continues to move in the same direction as the mirror, he may place a convex spherical lens before the eye so as to render it artificially myopic. Thus, if with a convex lens of 1 D. the shadow movement is annulled at a distance of 1 metre, the eye must be emmetropic, since one diopter of myopia is produced by a lens of 1 D. But if it requires a lens of 2 D. to bring the point of reversal to the one-metre distance, the eye must have 1 D. of hyperopia. In other words, if the examiner places himself at a distance of one metre from the eye, the hyperopia is always 1 D. less than the dioptric power of the lens which annuls the shadow-movement. In myopia of more than 1 D. the examiner may approach the eye until he reaches the point of reversal and thus measure the distance of the far-point directly, or he may (as is generally preferable) remain at the one-metre position and place before the eye such a concave lens as will bring the point of reversal to this position. The lens which effects this, neutralizes all but 1 D. of the existing myopia; that is, the myopia is 1 D. greater than the dioptric power of the lens which annuls the shadow-movement.

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[9] The movements as described are those that take place with a plane mirror; if a concave mirror is used, the direction of the movement is in all cases opposite to that described for the plane mirror.

If in the application of skiascopy we find (either with or without a spherical lens) no shadow-movement in one meridian, while a shadow appears to move across the pupil in the meridian at right angles to this, the existence of astigmia is demonstrated, and the meridian in which there is no movement is one principal meridian, while the other is the meridian in which the shadow appears to move. The degree of astigmia is then measured by the cylindrical lens, having its axis in the meridian of no movement, which, either alone or together with a spherical lens, annuls the movement in all meridians.

Keratometry, or ophthalmometry, consists in the measurement, by means of images reflected at the anterior surface of the cornea, of the curvature of this surface, and the deduction therefrom of the corneal asymmetry in terms of the dioptric equivalent of the resulting astigmia; but since the corneal astigmia is to a variable extent modified by the lenticular astigmia, which is not revealed by keratometry, this method is less valuable than skiascopy; it is, nevertheless, very useful as a corroborative test.

Determination of the Accommodative Power.—The accommodative power may be most conveniently measured by means of small test-letters constructed on the five-minute-angle principle, as have been devised by Oliver. If the eye is emmetropic, the accommodative power is measured by the shortest distance from the eye at which the smallest distinguishable letters can be read. If the eye is ametropic, its refractive error should be first corrected, and the examination then conducted as in emmetropia.

CHAPTER III.

THE PSYCHOLOGY OF THE VISUAL ACT AND THE FOCAL DISEASES OF THE VISUAL CORTEX.

BY CHARLES K. MILLS, M. D.

INTRODUCTORY OUTLINE.

The visual act may be described in general terms as that process by which luminosity, form or color is recognized by the organs of vision. Usually the act is concerned with the visual recognition of a definite object. The term has, however, an application to subjective visual processes as illustrated, for instance, by hallucinations of sight either in the normal or in the insane, and by the recognition of phosphenes due to mechanical or electrical stimulation. My concern in the present chapter is chiefly with the visual act as related to the cerebral cortex, and especially to the higher cortical areas and centres of vision. The consideration of the subject must necessarily include to a certain extent both the physiology and the pathology of the visual cortex. The cortical visual act may be simple or complex according to the simplicity or complexity of the object recognized, which may vary from a luminous point of weak intensity and brief duration through a series including forms of all grades of regularity or irregularity, colors differing in hues and in shades, natural objects of diverse sort, particular landscapes, persons with individuality of face and figure, designs originating in the arts and sciences, and numbers, letters and words.

Under the influence of the recent discovery of radium and its effects, some tendency is shown among physicists to return to the corpuscular theory of light; nevertheless the still most universally accepted doctrine is that which is based upon the Newtonian hypothesis of luminous transmission by waves of the ether which permeates all space.

Unlike sound vibrations, the direction of the waves of the vibrating ether is transverse to the direction of the rays of light. Only those ethereal vibrations which have a velocity of between four hundred billions on the one hand and a little more than nine hundred billions on the other hand are effective in giving rise to sensations of sight.

To others in this work are assigned the tasks of not only describing and explaining the anatomy and methods of action of the components of the eye itself, but also of many of the effects which result from the reception of light by the nervous apparatus of vision. I shall consider only the ultimate cerebral, and therefore psychical, effects of the light transmitted through the eye to the nervous system. In the retina are certain photo-chemical substances and a complex anatomical nervous structure, the chief constituents of which are known as the rods and cones. To and from these rods and cones come and go transmitting nervous structures which put the retinal elements in communication with lower and higher levels of the brain.

A light wave starts on its journey through the ether from the luminous object which is its source. The first step towards its becoming a visual impulse is taken when it decomposes the photo-chemical substances of the retina, thus setting up vibrations in the extreme periphery of the end organs of this membrane. The initial psychical step is taken when it reaches its first stopping place in the brain cortex.

The excitation received by the retinal substances and structures is conveyed by fibres of the optic nerve back to centres at the base of the brain, and either directly or by new relays of fibres to the visual cortex. Definite portions of the retina are related to equally definite portions of the visual centre which first received the projected retinal excitations—a matter which will later receive attention in more detail. The result in the cortical centres first receiving the impulses is a visual sensation or percept. Up to this point, however, an idea of the object perceived is not obtained. In order that this shall come to pass the cortical excitation which has been evoked must be transmitted to the cellular elements of another region of the brain surface; in other words, from a simple sensory or percept centre to a memory centre. A memorial image is deposited in the centre placed at some distance from the cell or cell-group in which the excitation is received.

When such focal diseases of the visual cortex as mind-blindness, word-blindness and letter-blindness are considered, the fact that visual sensations or percepts and visual concepts or apperceptions are dependent upon separate processes and have distinct cortical habitations, will more clearly appear.

One who is mind-blind or word-blind so that he may not be able to recognize persons or objects in their real characters, or words and letters in their true significance, may yet have visual sensations with their cortical correlatives, as illustrated by the fact that he can avoid or seek the objects which he does not recognize and that he may see the words and the letters composing them and yet not be able to tell what they import, although he knows that they are different from the other objects in his field of vision.

LIMITS AND SUBDIVISIONS OF THE VISUAL CORTEX.

I shall next define the limits and subdivisions of the visual cortex as I understand them. The visual cortex, that part of the convoluted cerebral surface concerned with the function of sight, includes the entire occipital lobe both on its mesotentorial and its lateral aspects; also taking in adjoining caudal portions of the parietal and temporal lobes. This cortical visual field is of complex composition. It includes a primary or lower and a secondary or higher cortical visual area. The higher area is extensively subdivided. The primary or lower cortical visual area which corresponds to the optic perception field of Wilbrand and Saenger[1], is situated chiefly, but not exclusively, on the mesotentorial surface of the occipital lobe; the secondary or higher cortical visual area corresponding to the optic memory field of the authorities just mentioned, covers a large extent of the lateral aspect of the occipital lobe, spreading, to some extent, over the temporal and tentorial surfaces.

In order to understand the limits and subdivisions of the visual cortical areas, in accordance with the broad statements just made, it is necessary to have some idea of the views of Flechsig.[2]

His views are based upon his studies in myelinization as especially observed in the brain of the fœtus and of the infant shortly after birth. He believes that the nerve fibres connected with special cortical areas become medullated at certain definite periods, these myelogenetic areas having special relation to the evolution of particular functions. Some of the areas appear at the same time, others at a time separated by weeks or months from the areas which have preceded them. Thirty-six of these areas have been marked out by Flechsig on the cerebral cortex.

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[1] Wilbrand and Saenger, Die Neurologie des Auges, vol. iii. Anatomie und Physiologie der Optischen Bahnen und Centren, Wiesbaden, 1904.

[2] Flechsig, Gehirn und Seele, ii. Aufl., Leipz., 1896, Taf. iv. Flechsig's views are presented in a condensed form in an article over his own signature in The Lancet, vol. ii. 1901, p. 1027.

The references somewhat frequently made to Wilbrand and Saenger, unless otherwise indicated, refer to the volume above noted. Much use has been made of this valuable book, which is especially rich in its summary of the work of German writers and investigators. In order not to overload the chapter by a repetition of references, I might here call attention to some of the most important monographs or treatises used in its preparation, although I cannot make this list complete. These include Kussmaul's Disturbances of Speech in Ziemssen's Cyclopædia of the Practice of Medicine, Amer. edition, vol. xiv., New York, 1877; Ferrier's Functions of the Brain, 1886, and Croonian Lectures on Cerebral Localization, 1890; Gowers's Diseases of the Nervous System, 1893, and his Clinical Lectures on Subjective Sensations of Sight and Sound, 1904; Wyllie's Disorders of Speech, 1894; and v. Monakow's Gehirnpathologie, Wien, 1897. Although it is giving scant justice to others, space will only permit me by naming them to acknowledge my great indebtedness to Hitzig, Munk, Horsley, Schäfer, Beevor, Saenger Brown, Dejerine, Henschen, Pick, Bateman, de Schweinitz, Swanzey, Oliver, and Elder, among many others. Besides this general acknowledgment, references are made in various parts of the chapter to particular contributions.

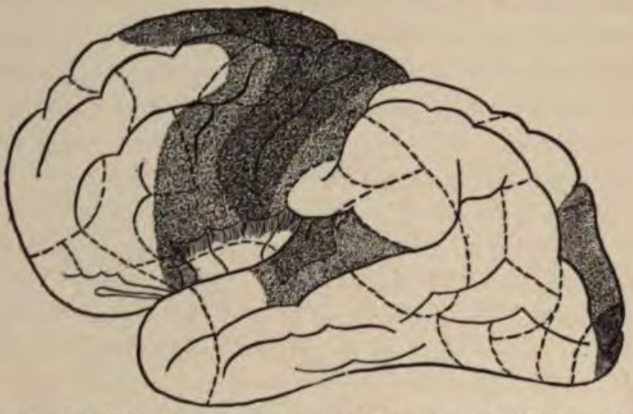

FIG. 1.—Flechsig's primordial and association areas on the lateral aspect of the human hemicerebrum; the primordial areas are shaded.

In this connection our chief concern is with Flechsig's great subdivisions of the cortex into: (1) primordial zones, and (2) association areas. The primordial zones correspond to the so-called projection areas, the regions of cortical representation of the special senses, common sensibility and of movements. The association areas occupy a large portion of the cerebral surface, nearly two-thirds in all. They are situated in those regions of the brain which were in times past often designated as latent simply because we did not know their functions. Instead of being functionless they are the seat of the highest and most special-

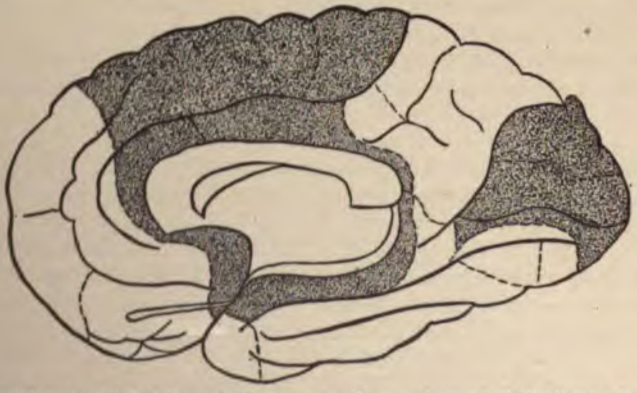

FIG. 2.—Flechsig's primordial and association areas on the mesal aspect of the human hemicerebrum; the primordial areas are shaded.

ized cerebral attributes. These association areas were divided by Flechsig into two, a posterior and an anterior. The posterior association area occupies both the lateral and the mesal aspects of the brain and includes portions of the parietal, temporal and occipital lobes. To it Flechsig also gave the name of intermediary area. The anterior association area which is situated exclusively in the prefrontal region has also been designated by Flechsig as the terminal zone. These primordial and association areas are represented diagrammatically in Fig. 1 and Fig. 2.

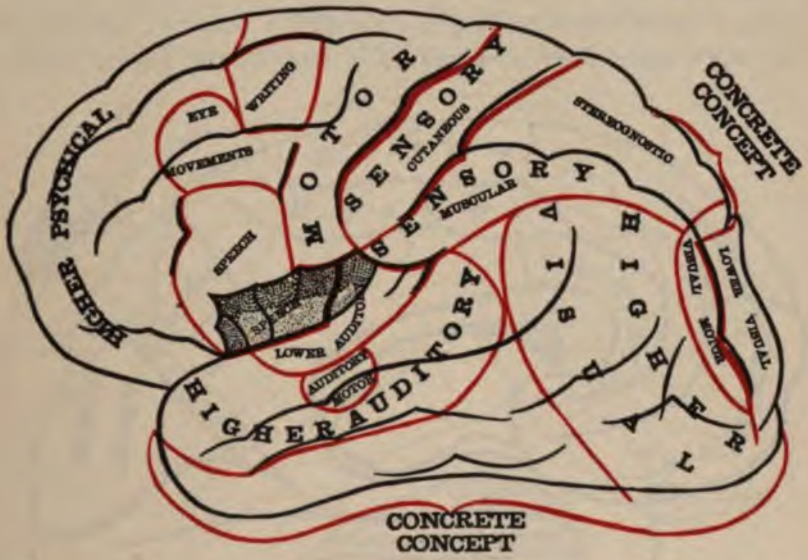

FIG. 3.—Diagrammatic scheme of the physiological areas and centres of the lateral aspect of the human hemicerebrum.

I have suggested that the term concrete concept area should be applied to Flechsig's posterior or intermediary area. It includes the optic memory field of Wilbrand and Saenger and much more, the memory field directly related to all of the senses; indeed, another designation which would be expressive of the functions of this great area would be the concrete memory field. This concrete concept area or concrete memory field is indicated in the diagrams, Figs. 3 and 4. This area is much subdivided according to the evolution of function. Tentatively its most important separate parts or centres might be indicated as represented in the diagram, Fig. 3. The views of Campbell, based on histological research regarding the physiological subdivisions of the cortex, are given with illustrations, See Figs. 11 and 12, pages 73 and 74. These views, arrived at by a different road, are similar to those of Flechsig and the writer.

Concrete concepts are of various kinds according to the degree of their simplicity or complexity. One may have a simple visual concept of a leaf or flower, made up purely of visual sensations and memories. This concept, however, becomes more complex, and at the same time more complete, when with it are associated the memorial images of the same flower or plant which are derived through other senses,—as those of touch and smell. This higher concrete concept can only be brought about by associations between visual and other sensory areas. Moreover, to the concrete concept, simple or complex, a name comes to be given through a series of cerebral processes which have their structural substrata in the higher auditory sphere; and to connect these with the visual areas, other associating tracts must be present. The highest psychical area of the brain, that concerned with attention and volition, with abstract concepts of every sort, is associated with the visual cortex, as it is with all other parts of the great concrete memory field; for it is only by such association that reason, judgment, and fancy can be brought to bear in connection with our concrete ideas.

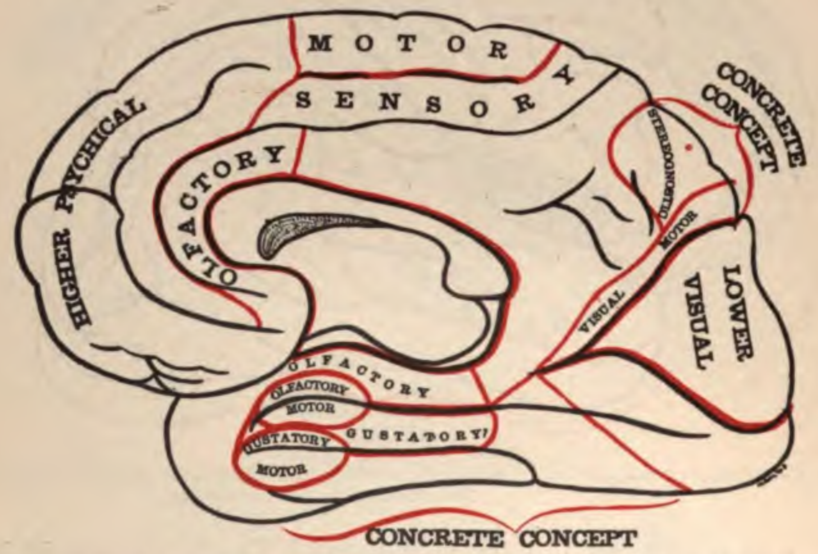

FIG. 4.—Diagrammatic scheme of the physiological areas and centres of the mesal aspect of the human hemicerebrum.

A few words might be said here regarding the differences between the lower and the higher visual areas in the lower animals and in man, in order to emphasize the manner in which the secondary or higher visual centres develop as evolution proceeds.

The lower or primary cortical visual area is largely developed in animals much below man which use their peripheral and central vision for some purposes even in a higher degree than man. They see acutely for long distances and have a large peripheral field. The optic memory field in animals below man and in savage races is highly developed as regards memories concerned with the recognition of those natural objects, places and persons, an accurate knowledge of which is necessary for them for defense and subsistence. The visual portion of the concrete memory field in the highly civilized human being is greatly enlarged and much elaborated in accordance with his acquirements.

The subdivisions given of the areas for concrete memories are of course provisional, and will doubtless be largely added to when our knowledge of the regional morphology, physiology and anatomy of the brain is increased. In Figs. 5 and 6 are shown the subdivisions of the visual areas on both the lateral and the mesoventral aspects of the hemisphere according to the writer's latest views.

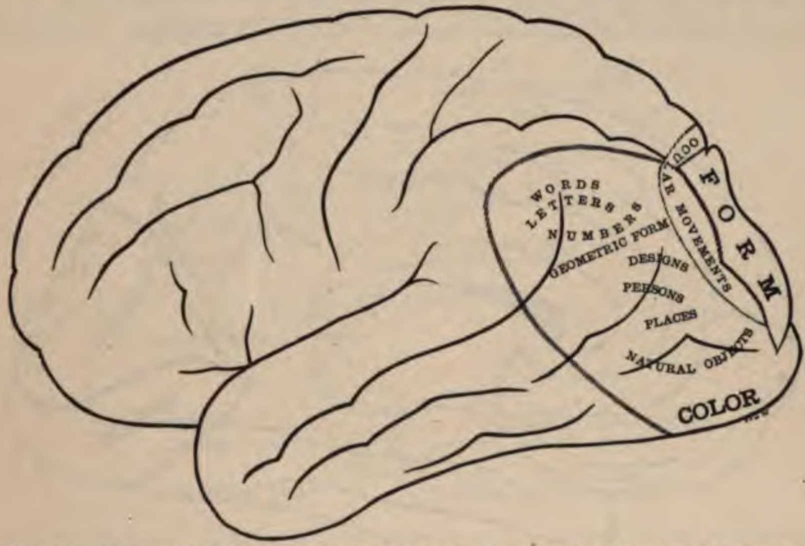

FIG. 5.—Scheme showing the extent and subdivisions of the secondary or higher visual cortex on the lateral aspect of the cerebral hemisphere.

It will now be necessary to speak of the connections and associations of the visual cortex. Between the calcarine and cuneal cortex and the pregeniculum and pulvinar of the thalamus runs a broad band of fibres long known as the optic radiations of Gratiolet, while the optic tracts connect the geniculate and thalamic visual centres with the retina. The fibres from the pulvinar run in the dorsal, those from the pregeniculum in the ventral section of these bundles. It is not improbable that in the visual radiations are fibres of at least two different origins, some fibres passing from the basal centres to the cortex and others from the cortex to these centres.

We shall not here go into details of the exact manner in which these connections from the retina and the occipital cortex are made, except to say that it is after such fashion that similar halves of the retina, of both eyes, are related to the occipital cortex on one side; the right halves of the retina to the right occipital cortex, and the left halves to the left occipital cortex; while a small central macular field has a bundle of its own which also makes connection with the cortex of the brain on both sides. Another connection of the retina through the optic tracts is with the anterior colliculus of the quadrigeminum, this having a path to the occipital lobe and others to the nuclei of the ocular muscles. The reader is referred to Dr. Spiller's article for a fuller consideration of the visual pathway. The entire retino-intracranial visual apparatus is well shown in the schematic diagram of von Monakow which is partly based on the work of Ramón y Cajal. The illustration is reproduced by permission of Professor von Monakow. I have modified it so as to introduce connections of the primary optic centres with the higher visual field. It is based upon von Monakow's experimental and pathologico-anatomical investigations.[3]

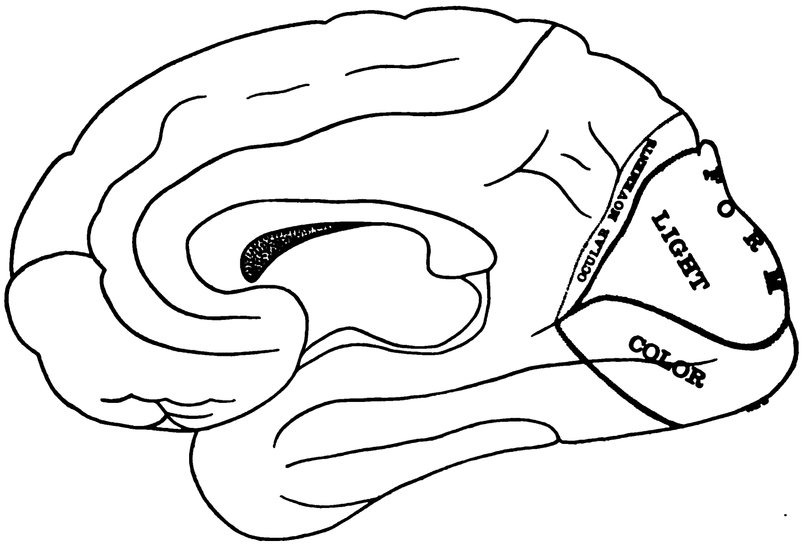

FIG. 6.—Scheme showing the position of the primary visual centre on the mesal aspect of the cerebral hemisphere; and also the probable positions of the centres for color recognition, for elementary forms, and for those ocular movements which are immediately responsive to visual impressions.

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[3] Von Monakow, Arch. f. Psych., vol. xx. 3, and Gehirnpathologie, 1897.

With the anatomy, physiology and diseases of the optic radiations, the basal optic centres, the optic tracts and the retina, I am not concerned in the present chapter. It is only necessary to recall here the fact that hemianopsia, of the most complete type, has its pathological seat in a lesion which severs the optic radiations or optic tract.

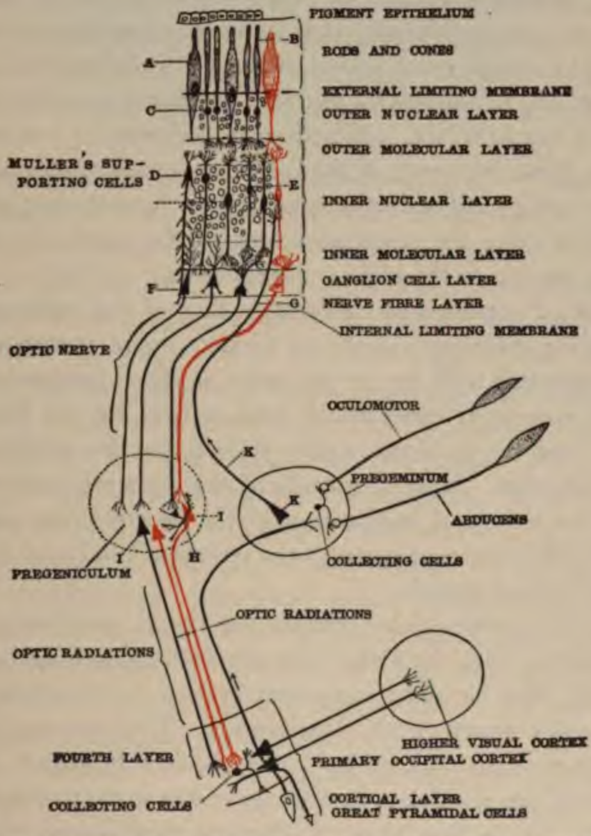

FIG. 7.—Scheme by von Monakow of the regions of the optic pathway (opticus) in the retina, and its further termination in the cortex of the occipital lobes. A, cones; B, rods; C, nuclei of the rods; D, bipolar ganglion cells for the cones; E, bipolar ganglion cells for the rods; F, large multipolar ganglion cells (cells of origin of the optic nerve); G, centrifugal fibres of the optic tract and nerve which originate in the pregeminum and end in the retina; H, reticular receiving cells of the pregeniculum: and I, the optic radiation cells of the pregeniculum. The course of the light waves towards the cortex is indicated by way of cones and rods; bipolar ganglion cells for the cones and rods (optic cells): receiving cells in the pregeniculum, optic radiation cells; cortical cells (third and fifth layer). In order to complete the cortical connections of the optic pathway an addition has been made to the diagram at H V, which indicates the higher visual cortex in the angulo-occipital region which is connected with the lower visual cortex by the lines running in the direction of the arrows. It must be remembered in studying this diagram that only the connections to one side of the occipital cortex are given. The semidecussation of the optic tracts is not represented. In addition, it must be recalled that tracts run from the primary visual cortex of each occipital lobe to the higher visual cortex of the opposite side. This also is not represented in the diagram, which is intended simply to give a clear conception of the connections of the retina with the basal ganglia and the primary visual cortex (primary optic centres).

Special sets of fibres associate the primary visual centres or optic perception fields of both hemicerebrums with the higher cortical centres of each side, as will be further discussed when the two clinical forms of word-blindness are considered. It is important that this double association should be borne in mind. This association is indicated diagrammatically in Fig. 18, in which O 1 and O 2 represent respectively the left and the right occipital lobe. According to Dejerine, it is brought about by the fibres from the cortex of the calcarine fissure of one side crossing through the forceps major in the splenium of the callosum, and joining with the fibres from the calcarine cortex of the other side, the united bundle then passing to the higher visual cortex.

A matter of considerable interest is that of the existence or non-existence in the *carrefour sensitif* of Charcot, or in juxtaposition to it, of a tract concerned with the transmission of visual impressions. Some of the older records of cases would seem to bear out the theory of the existence of such visual fibres in this position, but the weight of opinion is against this view. Most, if not all, of the cases are more readily explained on the theory of implication of the nearby optic radiations in the lesion which impairs or destroys the posterior bundle in the posterior limb of the internal capsule.

Besides the tracts which associate the visual perception centres of both hemispheres with the visual memory field of each side, other associating tracts must be taken into consideration in discussing the functions and focal diseases of the visual cortex. These associations are, in fact, so numerous that the subject would become befogged, at least for the purposes of the physician, if an attempt was made to discuss them in great detail. In the first place, the various subdivisions of the higher cortical visual field have more or less intimate associations with each other, in accordance with the degree of association between their functions. The centres in the sphere of object seeing which are concerned with the recognition of persons, places and natural objects are associated with the centres of language symbolization. The centres of the higher visual area generally are connected by associating tracts with similar concept centres in other portions of the concrete memory field, as, for instance, with the higher centres of hearing, touch, taste and smell.

HISTOLOGY OF THE VISUAL CORTEX, ESPECIALLY WITH REFERENCE TO VISUAL LOCALIZATION.

As in other parts of the cerebral cortex, histology throws some light upon localization. What is known as the layer of Gennari occupies a position which is supposed to indicate the extent and limitation of the primary visual cortex in man and the lower animals. The layer of Gennari spreads over the cortex in such a way as to include the lingual lobule from the junction of the calcarine and parieto-occipital fissures, caudad to the occipital pole, including the calcarine fissure, the entire cuneus, and the first occipital convolution. Fibres from the optic radiations have been traced to all the regions in which this layer is situated.

Space cannot be given to a full consideration of the minute anatomy of the visual cortex and its bearings upon the question of the development and the localization of cortical visual centres.

Meynert believed that in the occipital lobe the classic arrangement of the cortex into five layers, as especially seen in the motor region, was not followed. He recognized no less than eight layers. The present tendency is, following Ramón y Cajal, to recognize the same number of layers in the visual as in other portions of the cortex. Counting from without inwards, Cajal described five layers,—namely, (1) a molecular layer; (2) a layer of small fusiform vertical cells; (3) a layer of small pyramidal cells; (4) a layer of large pyramidal cells; and (5) a layer of polymorphous cells (Fig. 8). An acceptation of the views of Cajal brings the occipital lobe in harmony with other portions of the cortex, with primary functions like hearing, touch, smell, taste and the representation of movement. The studies of Cajal were made upon small mammals. The same layers modified by evolution are, however, recognizable in man and in the primates below man.

In Fig. 9 is shown a crossed section through the normal cortex of the left calcarine fissure in an eleven-year-old epileptic studied by von Monakow. The right hemisphere was the seat of a porencephalic defect.

Von Monakow's method of considering the visual cortex is exhibited in this and in other illustrations in his work. Regarding the subject in a purely topographical sense, the following layers according to him are recognizable in the cortex of the occipital lobes: (1) the ependymal and fusiform layer similar to the layers found elsewhere in the convolutions; (2) the layer of the small pyramidal cells, although even here there are a few examples of the large pyramidal cells; (3) the layer of giant pyramidal cells between, below, and above which are found accumulations of nuclei; (below the layer of the small pyramidal cells is the upper portion of the Vicq d'Azyr stripe and below this a collection of substantia gelatinosa—the lower part of the Vicq d'Azyr stripe extends into the fourth layer); (4) the fourth layer is constructed chiefly of polymorphous multipolar cells and contains many myelinated fibres; it is rich in ground substance; (5) this layer is constructed of elements similar to those of the fourth layer, but it is indicated as a separate layer because of the less number of horizontal nerve fibres which it contains.

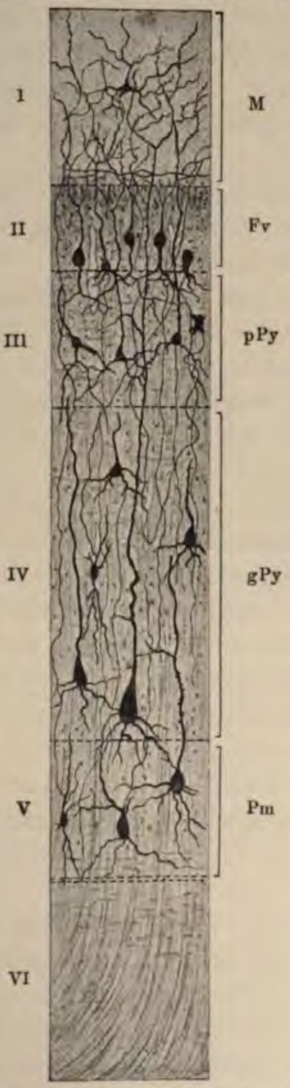

FIG. 8.—Scheme representing the layers of the occipital cortex according to Cajal; I, II, III, IV, V, are the five layers of Cajal; M, the molecular layer; Fv, the layer of fusiform vertical cellules; pPy, layer of the small pyramidal cells; gPy, layer of large pyramidal cells; and Pm, layer of polymorphous cells.

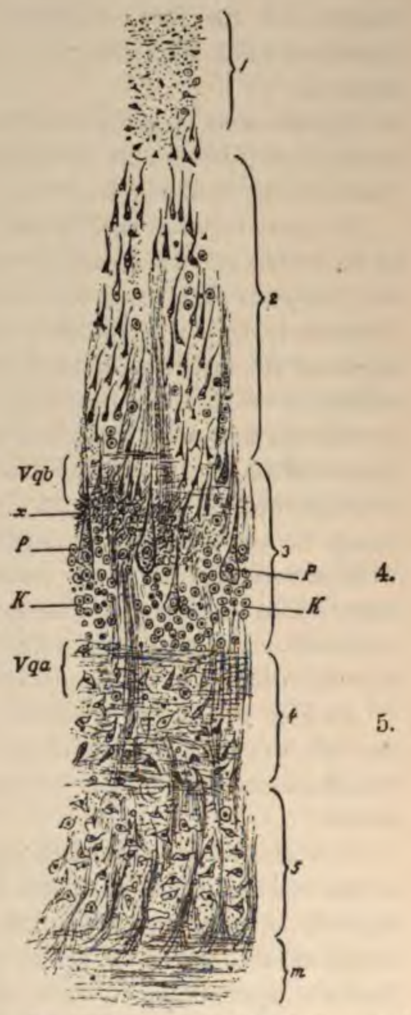

FIG. 9.—Crossed section through the normal cortex of the left calcarine fissure in an eleven-year-old epileptic with porencephalic defect in the right parieto-occipital lobes, and total primary destruction of the optic radiations on the right side, magnification 300. The five layers of the cortex are after Meynert,—K, nuclei; P, giant pyramidal cells; m, subcortical white matter. (von Monakow.)

A comparative histological study of the cortex of animals having vision, from the lowest to the highest, and a similar comparative study of the lower and higher visual cortical areas in man, bear out the views as to function based on other methods of investigation, as those of physiology and clinical pathology. Much work, extending over many years, has been done in this field of research, but the most valuable is without doubt that recently published by F. W. Mott and A. W. Campbell. The correlation of structure and function in the neuro-visual apparatus was demonstrated by Mott,[4] as exhibited in changing cell lamination of the visual cortex of mammalia from insects to the primates. The transition from monocular to binocular and from panoramic to stereoscopic vision is discussed, and the relation of such transition to full decussation and semi-decussation of the optic tracts is shown. The semi-decussation is correlated with "progressive development in the layer of the higher associational pyramidal cells lying above the layer of the granules in the cortex".

Mott illustrated the various stages of evolution of the visual cortex by actual preparations. He begins with a discussion of the extremely simple type of visual cortex shown by the mole and the shrew, with their limited visual powers. In these animals the cell structure of the cortex is of the most rudimentary character, consisting of small stellate or quadrilateral cells resembling granules, below which is a thin layer of polymorphic cells with a few large cells. These three types of cells, rudimentary in the mole and the shrew, are found throughout the series of animals studied by Mott. In rodents which have wide panoramic vision, but unimportant elaborations and associations, the visual cortex becomes considerably more complex.

In the rabbit is a higher elaboration of the visual cortex proportionate to the increased visual powers. Here are seen the visuo-sensory cells of Raymón y Cajal, a thin layer of large stellate cells, and closely packed pyramidal cells just above the granules. Up to this point in Mott's series the vision of the animal studied is chiefly panoramic or periscopic, being characterized but little by comprehension of depth and special form. Mott traces his series through the marsupials, ungulates, the canines and the felidæ, each mammalian form in the advancing series

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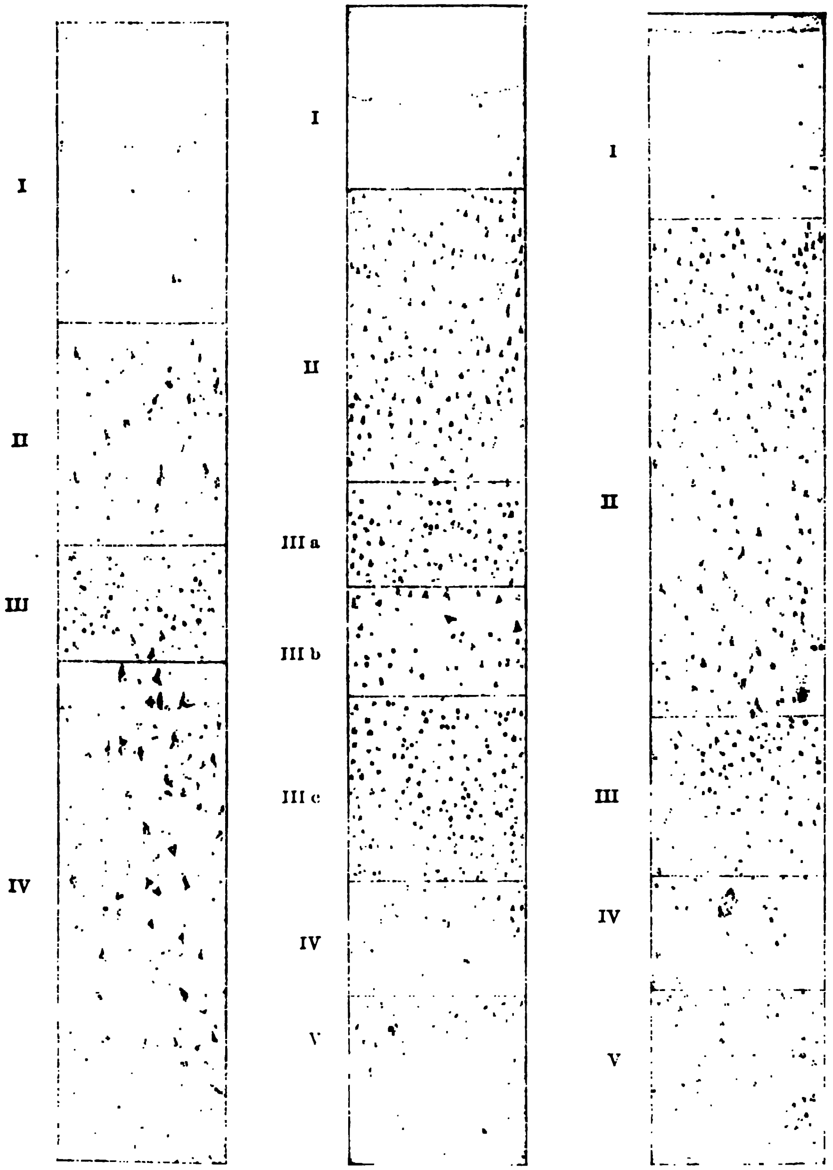
[4] Mott, The Lancet, vol. ii., December 3, 1904, p. 1555. Mott's results were presented in the Bowman Lecture, and are recorded in an authorized and carefully prepared abstract in the Lancet, from which the account here given is taken. I have reproduced, by permission of Dr. Mott, the illustrations given in Fig. 10, which represent two histological types of the visual cortex,—namely, in the mule-deer and macacus-monkey.



A—MULE-DEER. B—MONKEY. C—MONKEY.

FIG. 10.—Reproduction of drawings after photomicrographs of sections of the visual cortex in the mule-deer, and of the visuo-sensory and visuo-psychic cortex in the monkey (*macacus*). Magnification 150. The percentage of thickness of the different layers is only approximate.

A, Mule-deer, the visuo-sensory area: I, 20 per cent.; II, 20 per cent.; scattered pyramidal and large stellate cells; III, 11 per cent. granules; IV, 18 per cent. solitary cells of Meynert; polymorphic.

B, Monkey, the visuo-sensory area: I, 10 per cent.; II, 27 per cent. pyramidal; III a, 10 per cent. outer layer of granules; III b, 10 per cent. large stellate cells, line of Gennari; III c, 17 per cent. inner layer of granules; IV, 11 per cent. inner line of Baillarger, with solitary cell of Meynert; V, 15 per cent. polymorphic.

C, Monkey, the visuo-psychic area: I, 17 per cent.; II, 43 per cent. pyramidal; large pyramids; III, 14 per cent. granules; IV, 10 per cent. inner line of Baillarger, with solitary cell of Meynert; V, 16 per cent. polymorphic. (Mott.)

showing greater extent and elaboration of the visual cortex in accordance with the addition of new powers of vision, binocular and stereoscopic perception being added to uniocular and panoramic vision. The cat is probably the first animal in the series presented by Mott which is capable of convergent stereoscopic vision by movement of the eyes independent of the head. Below the cat complete decussation of the optic tracts is probably present. In this animal an imperfect semi-decussation, like that which is later seen in its fuller development in the primates and man, takes place. The cat is probably the first species in the series of mammals in which section of the optic tract produces hemianopsia. The visual cortex is developed proportionately in each species according to its increased powers. In the felidæ, to which the cat belongs, the pyramidal area is of increased depth, the solitary cells of Meynert very numerous, and the polymorphic layer diminished as compared with series lower in the scale.

Reaching the primates with their perfect binocular stereoscopic vision, which can be added to by movements of the eyes and head, a semi-decussation of the optic tracts, a distinct macula lutea, and a definite occipital lobe with the line of Gennari visible to the naked eye, are found. For the first time the visual cortex presents two structurally different regions, a visuo-sensory and a visuo-psychic. The visuo-sensory cortex attains its maximum extent and complexity in the ape and is characterized by increase of width of the line of Gennari, a double layer of granules, and above the outer of these a double layer of small and medium-sized pyramids. The visuo-psychic or associational cortex (not definite as a distinct zone below the ape) is characterized by an increased depth of the pyramidal layer owing to the presence of a third row of large associational pyramids, this region being progressive in extent upwards to man, in whom it attains its maximum development at a period subsequent to birth. The visuo-sensory and visuo-psychic cortical areas correspond to the primary and to the higher cortical visual areas respectively. The results of the researches of Campbell, which will next be referred to, are practically the same as those reached by Mott, although the investigations were in the main along different lines.

Campbell [5] has pursued a series of investigations on the medullated structure of the nerve fibres of the cerebral cortex, and also of the cortical cells, with the result that these investigations have served to show that such studies constitute a method of determining the functions of different regions of the cerebral surface as valuable as physiological investigations on the lower animals, embryological researches like those of Flechsig on the fetal brain, clinicopathological records of well observed cases, and morphological and anatomical studies of fissural and gyral development and arrangement. He has shown that histological studies of the form and arrangement of the nerve elements in the normal adult, the microscopical change present in pathological cases, and the relative histological findings in anthropoid apes and in man, all tend to one end—the demonstration and verification of the existence of specially arranged separate functional areas and centres. Among the clinicopathological cases those examined were instances of amyotrophic lateral sclerosis, of amputation of extremities, and of deprivation of the special senses. The results obtained are of the greatest interest, and in the main are confirmative of the views of Flechsig, of the morphological and anatomical studies of E. A. Spitzka and the writer, and of the clinicopathological facts accumulated during recent years by a large number of contributors to neurology.

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[5] Campbell, Proceedings of the Royal Society, vol. lxxii. Dec. 3, 1903; Journal of Mental Science, October, 1904.

"Viewed collectively," Campbell says, "the human brain harbors two varieties of centres, controlling what we may call 'primary' and 'higher evolutionary' functions respectively; the former are those common to all animals and essential to survival,—*viz.*, centres for movement and common and special sensation; the latter are those complex psychic functions in the possession of which man rises superior to all other beings."

Among Campbell's conclusions are (1) the motor region is cephalad of the central fissure; (2) an area for higher skilled movements, which Campbell calls the "intermediary precentral" area, just in advance of the motor area, corresponds practically to the so-called motor centres for speech and writing; (3) the prefrontal lobe, relatively weak in nerve cells and fibres, is the last pallium of the human brain, its functions as yet being uncertain; (4) the post-central convolution is the main terminus for common sensory impressions; (5) the post-central gyre is probably a primary area, one serving for the recognition of the simplest components in common sensation, as impressions of heat and pain; (6) just behind the post-central area is an intermediate post-sensory area concerned with the elaborations of sensations like muscular sensibility and stereognostic perception.

With regard to the visual cortex, with which I am here particularly concerned, the views of Campbell are in keeping with what has been indicated as his opinions regarding other portions of the cerebral surface; moreover, these views are in accord with those of Flechsig, the writer and others, who have endeavored to show that a large part of the parieto-temporo-occipital cortex should be regarded as a great concrete memory field, a region higher than those concerned with the mere reception of sensory impressions and the projection of impulses concerned with movements. Campbell believes, as the result of his studies, that the visual area is histologically divisible into two areas. The first of these, according to him, closely follows the calcarine fissure, extending only a short distance from it on each side. It is, he believes, intended for the reception of primary visual impulses. To it he gives the name visuo-sensory area. The second area skirts and invests the first, and is termed visuo-psychic, its purpose being the further elaboration of visual sensory impressions. It is characterized by a remarkable wealth of nerve fibres, coupled with the presence of curious large pyramidal cells. The various affections included under the general designation of psychic blindness are to be attributed to the destruction of this visuo-psychic area.

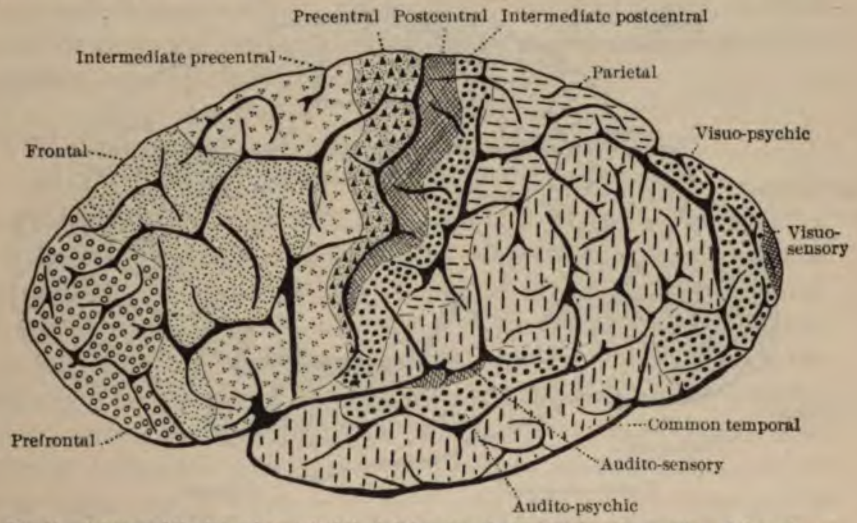

FIG. 11.—Scheme of the lateral aspect of the left hemicerebrum showing the position of the primary intermediate cortical functional areas. The primary or lower areas in the case of the special senses are designated, respectively, audito-sensory and audito-psychic. and visuo-sensory and visuo-psychic. The visuo-sensory area is present over a very limited field about the occipital pole, while the visuo-psychic spreads over a much larger area of the lateral occipital lobe. (A. W. Campbell.)

A study of the diagrams of Campbell (Figs. 11 and 12) shows that his visuo-sensory and visuo-psychic areas correspond in a general way to the subdivision of the visual cortex as given by me in this article. They differ somewhat, however, as regards the limitations and extensions of each of the areas. In accord with Henschen, he relates his primary or visuo-sensory area quite closely to the calcarine fissure. I believe it should be extended so as to include the whole of the cuneus, and possibly a narrow fringe of cortex on the lateral aspect of the occipital lobe. He restricts his visuo-psychic area to the occipital convolutions both on the mesotentorial and lateral aspects of the hemisphere, while I would extend it forward on the latter, so as to include the caudal extremities of the parietal and temporal convolutions. The principle, however, is the same.

In a general way the histological investigations of Campbell show that the posterior association area of Flechsig, the concrete concept area of the writer, is subdivided into sub-areas for the representation of concrete memories, those memories which are derived primarily by way of common sensibility and special sensation; they are, in other words, higher areas of vision, audition, common sensibility, olfaction and gustation.

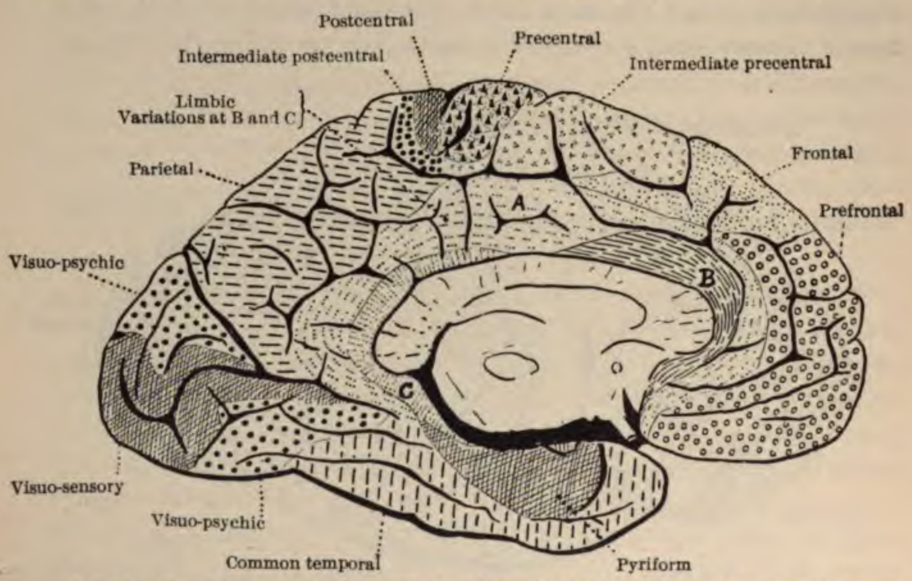

FIG. 12.—Scheme of the mesal and mesotentorial aspect of the left hemicerebrum. The visuo-sensory area as here indicated follows closely the calcarine fissure, while the visuo-psychic extends beyond the calcarine cortex in both directions. The various cortical subdivisions are indicated with clearness both graphically and descriptively. (A. W. Campbell.)

One point stands out prominently in Mott's investigations,—namely, that as vision advances increase in the development of the pyramidal cells takes place. Both in the race and in the individual development of the pyramidal cells, in number and in size, increases step by step with the evolution of power. Mott refers to the well-known myelination views of Flechsig, especially with reference to the optic radiations and the development of the different layers of the visual cortex. The intermediate zones of Campbell are but other names for the association areas of Flechsig or for the concrete concept or concrete memory field of the writer. In the visual cortex the intermediate visuo-psychic area is the optic memory field of Wilbrand and Saenger, the region already discussed as the seat of those higher visual powers such as the recognition of persons, places, objects, designs, and the symbols of language.

In his discussion of the development, structure, and function of the visual cortex, Mott refers to the morphological and anatomical studies on the brains of individuals of low and high development, and of those of different races, as of the Soudanese and Fellaheen, also of the Chinese, Goanese, Congo negro and the Egyptian. The work of E. A. Spitzka on the brains of men distinguished for their intellectual powers, and that of the writer on the brains of paranoiacs, imbeciles, negroes and criminals bear out the views of Mott.

POSITION AND THE SUBDIVISIONS OF THE PRIMARY CORTICAL VISUAL AREA.

The subdivision of the visual cortex into a primary or lower and a secondary or higher visual region having been considered, and such subdivision having been seen to be in accord with the views of Flechsig and the similar, although otherwise expressed, views of Mott and Campbell as to primordial and association areas, in the further discussion of our subject it is next necessary to glance at the question of the exact position, and the extent and subdivision of the primary cortical visual area. While all authorities are in practical accord in placing the primary or lower visual area, in whole or in very large part, on the mesotentorial surface of the hemicerebrum, they differ considerably with regard to the exact limitations and extensions of this physiological region.[6]

In studying the literature of the subject one is first struck with the great differences between authorities with regard to the extent of the primary cortical area. The extreme on one side is represented by the view of Henschen,[7] that the entire optic perception field is confined to the calcarine fissure, and the hypothesis is based on the fact that in one of his own cases of hemianopsia the lesion was really or apparently restricted to this fissure; and on the other hand by the view of Vialet,[8] that the primary visual area of the cortex includes the entire mesotentorial surface of the occipital lobe. Seguin [9] confined the primary centres for vision to the cuneus. Not a few of the physiologists and clinicopathologists who have written on the subject have included not only the entire mesotentorial occipital cortex, but also a large part of the lateral occipital lobe, and some like Ferrier have even extended the region into the temporal and parietal lobes. It must be borne in mind, however, that some of these writers have not made the distinction which is drawn here and should always be made between the primary and the secondary visual regions of the cortex. Not a little confusion has arisen, especially in the earlier consideration of the subject, by a non-recognition of this distinction. The only truly primary or primitive visual function is the recognition of luminosity, or perhaps it would be better to say the recognition of lightness or darkness. Color recognition, and that of form, come later in evolution. In a strict sense, therefore, the most primitive centres for vision are those concerned only with the appreciation of lightness and darkness. I believe that the centres for luminosity, color and form are distinct in the cerebral cortex. The color centres and the centres for form have developed out of the centres for light, just as the ability to recognize color and form has developed upon the basis of luminous recognition. This subject will be more fully treated when the cortical centres for color are discussed. It will only be necessary for clearness to state here, that it is view of the writer that the centres for color are contiguous to the primitive centres for sight, and that it is probable that they are located in the lingual and fusiform lobules below the calcarine fissure, while the centres for form, beginning at the mesal edge of the occipital portion of the hemisphere, are spread out in more and more complex development on the lateral aspect of the hemicerebrum chiefly in the occipital convexity. The primary centre for vision as here defined is, I believe, situated in the cuneus, the calcarine fissure and the cuneal slope of the parieto-occipital fissure.

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[6] Dr. Wm. G. Spiller, in his chapter in this work on the Intracranial Portion of the Encephalic Nerves, Especially of Those Related to the Eye, in the section on Primary Optic Centres, goes at some length into the question of their position, and the reader is referred to this portion of his article in connection with what is here said. It is necessary, however, to completeness, and in order that the exact views of the writer with regard to cortical physiology of vision may be understood, that he should here give some consideration to the location and extent of the primary cortical centres for sight.

[7] Henschen, Klin. u. anat. Beitr. z. Pathol. des Gehirns, Upsala, 1890–1903.

This primitive cortical area for sight, located in the cuneus and the calcarine and parieto-occipital fissures, has in the first place, two subdivisions, a peripheral and a central. The central subdivision is for the cortical representation of the macula and the peripheral for the rest of the retina. In the affection known as hemianopsia frequently the blotting out of the visual field is not, strictly speaking, as the name indicates, for exactly half of this field but for one-half of it less a small semicircular area which is inserted notch-like into the hemianopsic area at its internal central part. As Swanzey [10] expresses it this small area of retained vision is bitten out of the blind field. A patient may have double hemianopsia as the result of successive attacks involving both cerebral hemispheres in the occipital region, and circumscribed central vision remain.

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[8] Vialet, Les centres cerebraux de la vision, etc., Paris, 1893.

[9] Seguin, Jour. Nerv. and Ment. Dis., vol. xiv., 1887.

These and other facts would seem clearly to point to separate regions of representation for the central and for peripheral portions of the retinal field respectively. As will more fully appear later, I hold that both in the lower and in the higher visual fields, the macula and the peripheral retina are separately represented, but first let me speak of the probable position of the separate centres for the macula and periphery in the primary cortical visual area. For the present I believe it is best to accept the view of Henschen which has his own well recorded case in its support, and a few other facts, that the centre for the macula in the primary visual cortex is situated in the more anterior portion of the calcarine fissure, that is towards the apex of the cuneus.

Tentatively it may also be assumed that a horizontal zonular strip of the retina is represented in the posterior part of the calcarine fissure. These two localizations are based upon cases of hemianopsia with lesion of the middle portion of the calcarine fissure and adjoining cuneus, while the frontal and posterior parts have escaped.

Cases of cortical disease, quadrant and sector hemianopsia, and cases with ring-like or other scotomata in the visual field, are on record. Such cases are only to be explained on the supposition of a subdivision of retinal representation in the visual half centres. Much difference of opinion has arisen as to how this subdivision takes place, and the cases reported in proof of various positions are somewhat confusing and contradictory. A case of Henschen, for example, would seem to show that the upper part of the retina is represented in the superior lip of the calcarine fissure and the lower part in the inferior lip, and that a horizontal strip between the upper and lower parts of the retina is represented at the posterior extremity of this fissure; but the weight of facts is certainly against the view that the entire half vision centre is situated in the depth of the calcarine fissure. If this were true, in a case recorded by Beevor and Collier,[11] left hemianopsia should have been present, or at least left hemianopsia with the exception of a small area around the fixation point. The calcarine fissure, the inferior portion of the cuneus dorsal to it and the lingual lobule on its ventral side, were all destroyed, yet quadrantic hemianopsia alone, the fixation point escaping, was present. The only explanations which suggest themselves for Henschen's case are that in some persons retinal half representation is more concentrated cortically than in others, or as suggested by Beevor and Collier, the hemianopsia was due to involvement of the optic radiations in addition to the calcarine cortex. The case of Beevor and Collier is referred to in some detail by Spiller, see page 9, and will again be recalled when I consider cortical anopsias and hemianopsias. I shall only say here that it seemed to demonstrate that the lower quadrant of the visual field is represented by the upper or dorsal portion of the cuneus, as in their case the patient presented blindness of the left upper quadrant of the visual field, the fixation point escaping.

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[10] Swanzey, Eye Disease and Eye Symptoms in Their Relation to Organic Diseases of the Brain and Spinal Cord, in System of Diseases of the Eye, edited by Norris and Oliver, Philadelphia, 1900.

The case of Hun [12] has long held an important place with regard to this question of the subdivision of retinal representation. In this case the defect of vision was in the lower left quadrant of each eye, and the lesion as recorded by Hun was atrophy of the lower half of the right cuneus. Hun argued from this case that the representation of the lower quadrant of the visual field was in the lower half of the cuneus. I have re-examined Hun's original report of this case, and am inclined to agree with Beevor and Collier and others in the view that the case does not fully support the contention that the lower half of the cuneus is the area of representation for the lower quadrants of the retina of the same side. It is notable in the record of the case that the necropsy was made thirteen days after death and after the body had been embalmed, and also that Hun reports that the subcortical white matter was involved in the lesion to the depth of at least one-third of an inch. Probably as no microscopical examination was made, and as a brain lesion usually extends beyond the point where it can be traced by the naked eye, the degree of involvement of the subcortex and therefore of the optic radiations, was greater even than would be understood by the statement that the destruction extended to the depth of one-third of an inch. The case therefore has less value in the study of the subdivisions of retinal half representation than that of Beevor and Collier. In their case the lesion was studied with extreme care both macroscopically and microscopically, and showed that the cortex alone was involved.

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[11] Beevor and Collier, Brain, part 2, 1904, p. 153.

[12] Hun, Amer. Jour. of the Med. Sci., vol. xciii. Jan.–April, 1887, p. 140.

My conclusions with regard to the subdivisions of retinal representation in the primary cortical visual centres are: (1) that a lower quadrant of the field, or a little less than a quadrant, is represented in the upper half or two-thirds of the cuneus; (2) that the upper quadrant or a little less than a quadrant, is represented in the lower half of the cuneus, including the middle portion of the calcarine fissure; (3) that the macula, or at least the fovea, is represented in the anterior extremity of the calcarine fissure and its cuneal border; and (4) that a zone or strip of the peripheral retina is represented in the posterior extremity of the calcarine fissure and its cuneal border. When I speak of a little less than a quadrant being represented, it is because the strip supposed to be represented in the posterior limits of the calcarine fissure passes horizontally from about the line of the fixation point through the peripheral retinal field. It will be seen that in coming to these conclusions I have adopted in part the views of Henschen and in part those of Beevor and Collier. Doubtless the differentiation of retinal representation in the primary visual cortex is much greater than is here indicated, but the facts at command only permit at present this degree of subdivision.

FOCAL DISEASES OF THE VISUAL CORTEX.

The focal diseases of the visual cortex are those disorders of sight and intelligence which result from lesion or disturbance of any part of the lower or higher visual zones. The most important of these affections and those which will be considered in the present chapter are: (1) cortical anopsias and hemianopsias; (2) hemiachromatopsia and other forms of color-blindness of cortical origin; (3) central amblyopias crossed and uncrossed; (4) mind-blindness, total or partial, and transient or permanent; (5) word-blindness, letter-blindness and number-blindness; and (6) optic aphasia. Various disorders dependent upon visual cortical disease or related to it, such as alexia, paralexia, dyslexia, visual agraphia, paragraphia, and dysgraphia, will receive consideration, as will also the cortical and transcortical mechanism of visual spectra and auræ.

HEMIANOPSIAS AND OTHER DEFECTS IN THE VISUAL FIELDS DUE TO CORTICAL LESION.

It will not be necessary to go at length into a consideration of the first of the classes of focal disease due to lesions of the visual cortex, the hemianopsias and the other more restricted defects. The subject has already necessarily received some consideration when discussing the position, limitations and subdivisions of the primary cortical visual area, and it has also claimed the attention of Dr. Spiller in his discussion of the primary optic centres. The unusually interesting cases of Hun and of Beevor and Collier have been given, and some reference has been made to other cases.

A word or two concerning the terms most commonly used in describing the diseases of the primary cortical areas may serve to keep the subject well in hand. In the first place it should be remembered that hemianopsia, of whatever type, may be partial or complete. It may be partial as regards the amount of the visual field obscured or with reference to the degree of this obscuration. An absolutely total hemianopsia is one in which the dividing line between the blind and the unimpaired field passes through the fixation point. In practice, however, it is customary to describe those cases as hemianopsic in which there is a small central area of retained vision, a fact which has been referred to several times. In quadrant anopsia only one-half of the half-field is obscured. A few almost typical cases of this form of impairment of the visual field have been recorded. In some cases, however, a little less than a quadrant of the entire visual field is lost, a ribbon-like area of retained vision being left at about the position of the horizontal meridian. In double hemianopsia, as the result of simultaneous or successive attacks of homonymous lateral hemianopsia, the entire field of vision usually, with the exception of a small central area, is obscured. By a relative anopsia or hemianopsia is meant a partial impairment of the power of visual recognition in a portion or in one-half of the visual field. This relative hemianopsia may take several forms. The patient may, for example, in the impaired field recognize the color, form, and luminosity of an object, but not with the same clearness and intensity as in the unimpaired field. Hemiachromatopsia is, in a sense, a relative hemianopsia; the patient, while not recognizing colors in the affected field, is able to recognize form and light. (See Chap. IV, p. 167.)

Everyone familiar with the gross and microscopic pathological anatomy of the brain knows that it is extremely rare to find disease strictly limited to the cortical cinerea. Hemorrhagic lesions almost invariably involve both cortex and subcortex, and often the inner membranes as well. The same remark applies with increased force to abscesses of the brain; and tumors are almost never limited to the cortex, although growing, as they frequently do, from the membranes, they may for a time simply erode or displace it, later causing destruction of both cortical and subcortical substance. Softening due to either embolic or thrombotic occlusion, while it often includes a large cortico-subcortical area, may be limited to either the subcortex or the cortex. What closure of a vessel will cause is, of course, conditioned by the parts supplied by it. In rare cases a cerebral hemorrhage involves so little of the subcortex as to make it practically a cortical lesion. From the above statements it will be seen that the classes of cases which throw most light upon the exact functions of cortical visual areas, as of other regions of the brain surface, are tumors growing from the membranes before they have invaded deeply the cerebral substance, and a few cases of necrosis restricted to the cortex. The cases of Hun and of Beevor and Collier were of the latter class.

Cases of purely cortical or of nearly purely cortical hemianopsias or anopsias are extremely rare. A few have been recorded and are to be found in the most recent works on ophthalmology and neurology. Wilbrand and Saenger note the most important of these. In one case left-sided hemianopsia was present, a small portion of the visual field just beyond the fovea, three to six degrees, not being obscured. Necropsy and microscopical examination showed an exclusively cortical softening in the depths of the calcarine fissure. (Holm.) In another case of complete right-sided homonymous hemianopsia, the necropsy revealed calcarine softening. (Hilden.) Another case of permanent hemianopsia with insular formed scotomata in the neighborhood of the horizontal meridian in which a cortical focus of softening was found in the floor of the calcarine fissure has been recorded. (Eggers.) In addition to these cases of what appears to have been strictly cortical lesion, a few others have been reported in which the disease extended very slightly into the subcortex.

It sometimes happens that a lesion of the cortex of the lateral occipital lobe apparently causes a true hemianopsia, but in these cases the local pathological etiology is only apparent. A closer examination always shows that the lesion involves the optic radiations somewhere in their course from the pregeniculum to the calcarine fissure and cuneus. This is the explanation of a number of cases of hemianopsia which have been recorded as occurring in connection with lesions of the angular gyre and other parts of the lateral temporo-occipital region. Von Monakow gives an illustration of a case of this kind, the surface appearance being shown in Fig. 13. In this case, by making frontal sections through and cephalad of the lesion so as to expose the cortex, corona radiata and other related parts, it was found that the original lesion involved to some extent the optic radiations, in which also secondary degeneration had occurred. Von Monakow gives illustrations of the hemianopsic fields.

Double hemianopsia is of comparatively infrequent occurrence, but a sufficient number of cases have been recorded, both with and without necropsy, to shed some light upon its pathology in different cases. In most of the instances of this affection with necropsy both cortex and subcortex have been implicated in the lesion. It is indeed the rule in any case of hemianopsia when the cortex is involved for the subcortex also to take part in the disease, but the reverse is not so common. In other words, lesions of the optic radiations, the cortex remaining free, are relatively frequent and of course, in those cases in which the hemianopsia is due to lesion of the optic nerves, tracts, or pregeniculæ, the question of cortical implication does not arise.

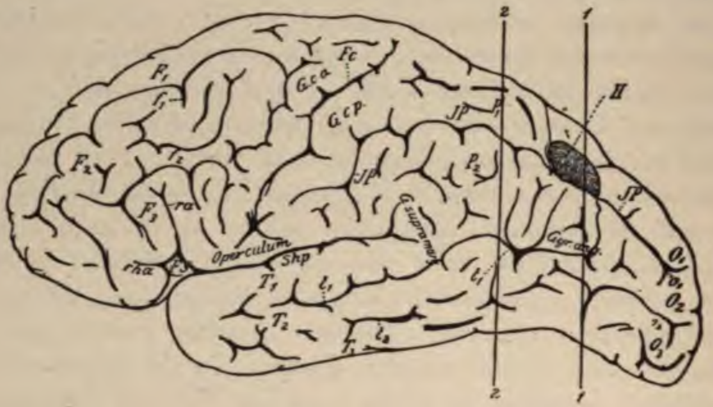

Fig. 13.—Lateral aspect of the left cerebral hemisphere in a case of apparent cortical hemianopsia. *H*, lesion in the interparietal fissure, and also of the superior parietal convolution (*P*, *i*), which is in large part destroyed. The line 1-1 represents a transsection made through the hemisphere and the posterior portion of the lesion. The line 2-2 indicates the position at which a transsection was made a short distance in front of the lesion. It is so placed as to about pass through the angular gyre. These sections were made in order to study the relation of the lesion to the cortex and subcortex. The optic radiations were involved in the lesion in which also secondary degeneration had occurred. (Von Monakow.)

In discussing double hemianopsias the fact already alluded to must be emphasized, that in lateral homonymous hemianopsia the loss sometimes extends over the entire visual half-field to a dividing line which passes through the fixation point, while in other cases a small central area of retained vision near the fixation point is left. In double hemianopsia two similar sets of cases are likewise observed; both halves of the field may be blotted out to the dividing line, causing, of course, complete blindness, or a small central area on each side of the fixation point may be left, leaving a form of tubal or barrel vision. In most cases of double hemianopsia one-half of the visual field is obscured, and later the other. In other words, a lateral homonymous hemianopsia becomes, as the result of successive attacks, converted into a double hemianopsia, either complete or with a small area of vision representing the fovea and its immediate environment. As already intimated, the cases of hemianopsia, single or double, with a retained area of central vision, are instructive in connection with the question of the existence of a separate macular centre in the lower cortical visual zone. The cases can scarcely be explained except on this supposition.

With regard to double hemianopsia of cortical origin, with which I am particularly concerned, cases with lesions strictly limited to the cortex can scarcely be found in literature, although in some of the few cases recorded the lesions were largely cortical. The case recorded by Dejerine and Vialet [13] had several points of particular interest. The double hemianopsia, or rather the complete loss of vision, took place at once and without any recognizable apoplectiform attack. Peripheral and basal disease were eliminated and the diagnosis of cortical blindness was made. The patient died after many years and necropsy showed bilateral lesions. On the right the lesion involved the cuneus and the lingual and fusiform lobules; in fact, the entire inferior temporo-occipital lobe; on the left the cuneus escaped, but the lingual lobule and other portions of the temporo-occipital lobe corresponding to the seat of disease on the right were included in the lesion. It is probable that the calcarine fissure was implicated. In all cases of double hemianopsia in which cortical lesions have been present, these have been situated on the mesoventral surface of the occipital lobe, and, as far as I know, the calcarine fissure has not escaped in any. The cases recorded with necropsy besides that of Dejerine and Vialet, just referred to, are those of Schmidt-Rimpler [14] and of Bouveret.[15]

I was consulted with regard to the interesting case which has been recorded by Dunn [16] and which is now part of the literature of the subject of double hemianopsia. In this case the patient had loss of power of locating or orientating himself. He could not form a conception of the geography of his house nor of any other place to which he had ever been. He could remember a place by its name, but could not call it up and relate it with its environment. He retained, however, the ability to recall and to describe individuals. A small central area of retained vision remained. From a study of his case, Dunn suggested the existence in the brain of a geographical centre, where are recorded the optical images of locality. It will be noted that in one of my schemes of cerebral zones and centres I have placed a centre for orientation along the anterior border of the inferior portion of the visual field.

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[13] Dejerine and Vialet, Centralblatt für Augenheilkunde, February, 1894, S. 64.

[14] Schmidt-Rimpler, Archiv für Ophthalmologie, November, 1887.

[15] Bouveret, Revue générale d'Ophtalmologie, November, 1887.

[16] Dunn, University Medical Magazine, Philadelphia, May, 1895.

Cases like that of Dunn and a few others which have been reported appear to indicate that, in order that loss of orientation shall occur, the lesions must be bilateral, that both the right and the left occipital or temporo-occipital regions must be the seat of destructive lesions. This is not, however, strictly correct. Cases are on record in which a destructive lesion of one hemicerebrum caused loss or impairment of orientating power. Such a case has been put on record by Peters.[17] The lesion was right-sided, the patient having left lateral homonymous hemianopsia. A lesion situated on either the right or the left side is apparently competent to cause impairment or loss of the power of orientation, as in another case, that of Groenouw,[18] the hemianopsia was for the right fields. It would seem to be much the same with disorder of orientation as with mind-blindness, of which, perhaps, it is an expression,—namely, that in the most usual case and most decided form it is the result of bilateral lesions, but may occur in special instances, either transiently or permanently, from a lesion of one hemisphere.

Relative cortical hemianopsia is a comparatively rare affection. If the theory of Wilbrand were true that light and color are represented in superimposed strata, some forms of it would be explicable on the view that the most superficial layers of the cortex were destroyed. Such destruction might, for instance, cause a hemiachromatopsia, leaving light perception unaffected, but it is fanciful, if not absurd, to reason that any such neatly demarcated destruction of superimposed cortical layers takes place as the result of disease, and, so far as I know, such a case has never been reported. The manner of blood supply to the cortex and subcortex would also make such a lesion unlikely. On the theoretical grounds advanced by Wilbrand, that form of relative hemianopsia which is known as hemiachromatopsia might be explained, but the explanation is not tenable. In the next section the probable lesions causing hemiachromatopsia will be considered.

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[17] Peters, Archiv für Augenheilkunde, xxvii. S. 175.

[18] Groenouw, Archiv für Psychiatrie und Nervenkrankheiten, xxiii. 339.

Relative hemianopsia sometimes takes the form of a diminution of the power of recognizing light, form, and color in the affected field, as in a recent case studied by me in which the patient had the general symptoms of brain tumor, such as headache, double optic neuritis, and vertigo, with other focal symptoms, besides lateral relative hemianopsia pointing to the left occipital lobe and especially its mesoventral surface. In this case the relative hemianopsia eventually became complete. A relative quadrantic anopsia may be present, and what is still more interesting, the case may show in one part of the obscured field complete loss of the powers of recognizing light, form, and color, while in another the loss may be only for color or form. Of course, if the power of recognizing lightness and darkness is lost, color and form recognition go with it, but form or color appreciation, or both of these, may be abolished and light and dark recognition remain. It may be that eventually some clearer insight into the exact position of separate centres for light, form, and color will be obtained by a study of cases in which there is loss of the three senses in one part of the field, and of only one or two in the other. Some curious forms of relative achromatopsia will be referred to, in the section on the color sense and cortical color centres.

In the differentiation of cortical anopsias and hemianopsias, as well as hemianopsias due to lesions of the optic radiations of Gratiolet, the importance of the Wernicke symptom, inaction of the pupils on stimulation of the blind half of the retina must be remembered. It is only necessary to recall that in cortical, as in optic radiation cases, the pupillary response remains for the entire retina, while in cases of disease of the optic nerves and tracts and of those portions of the basal ganglia which are concerned with vision, the pupillary inaction can be demonstrated. Some apparently conflicting cases have been recorded, but these are probably due to imperfect observation. It is well known to experienced ophthalmologists and neurologists that it is one of the most difficult of undertakings to demonstrate with certainty Wernicke's hemianopic pupillary inaction, but nevertheless it can be done if sufficient care is taken. In two cases in which Ferrier accidentally cut the optic tract in the course of his experiments on monkeys, the pupil failed entirely to respond to a pencil of electric light carefully focused upon the blind half of the retina, while in a number of cases of cerebral extirpation experiments the pupil never failed to respond.

The Wernicke inaction symptom, however, only serves to place the lesion in the cortex or the optic radiations. In determining whether the lesion is in the optic radiations or in the calcarine cortex or cuneus, the diagnosis must be reached by a study of the associated symptoms and of some special features of the hemianopsia. Hemianopsia due to lesion of the optic radiations is only in extremely rare cases an isolated symptom. Its associated manifestations may be few or comparatively many. It may, for instance, be part of a symptom complex which includes word-blindness, mind-blindness, and word-deafness, with other symptoms so often combined with these forms of sensory aphasia, such as alexia, dyslexia, and paraphasia. If the lesion is deeply placed so as to implicate the posterior portions of the posterior half of the internal capsule, hemianæsthesia may be present, as may also impairment or loss of the muscular sense and stereognostic perception. When hemiplegia or hemiparesis occurs with hemianopsia, which can be referred to the optic radiations, the lesion is usually an extensive one, although so-called distant symptoms may be exhibited.

The foregoing are some of the associations by means of which one is enabled, with some approach to certainty, to conclude that the lesion causing the hemianopsia is subcortical, and probably situated beneath and in particular relation with special portions of the lateral aspects of the postparietal and temporo-occipital regions. Hemianopsias, cortical, or largely so, such as were illustrated by the cases of Henschen, Holm, Hun, Beevor and Collier, are usually distinguished by the absence of such symptoms as hemianæsthesia and the various symptoms classed under the general head of visual and auditory aphasias; but if both cortex and subcortex are involved, the associated clinical phenomena will depend upon the depth and extent of the lesion. Quadrant anopsia suggests a cortical origin, although not necessarily, as it is altogether probable that dorsal, ventral, and medial portions of the retinal half-field are represented by separate bundles in the optic radiations, as they are by subdivision of the cortex in the cuneus and calcarine fissure.

Dufour[19] has suggested a special means of separating cortical hemianopsia from the same symptom dependent upon disease of the optic radiations. If the case is purely cortical, he holds that the patient will suffer from "vision nulle," that is, from the absence or negation of vision. The patient is not conscious that he cannot see in the blind field until his attention is called to the fact by perimetric or other investigation, or by some accidental circumstance. If the hemianopsia is due to lesion of the optic radiations, on the other hand, he has a subjective sense of darkness. It is doubtful whether this is a reliable test of cortical anopsia and hemianopsia, as a few cases have been recorded which seem to be in contradiction to Dufour's views with regard to negative vision; in other words, in cases in which the necropsy demonstrated the existence of a lesion of the optic radiations, Dufour's negative vision was observed.

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[19] Dufour, Revue médicale de la Suisse Romande, August 20, 1889.

Before taking up the subject of cortical color representation and chromatic disorders due to disturbance or disease of color centres, I shall here make a few suggestions as to terminology which seem to me to have some practical value. Hitherto in the discussion of the diseases of the cortical half-vision centres I have made use of the term hemianopsia or anopsia. It might be better, recognizing that the cortical representation of light, form, and color is separate, to give distinct names to the three forms of hemianopsia,—the visual half loss for light, for form, and for color. The terms achromatopsia and hemiachromatopsia are well-grounded in medical literature. To express the loss of the light sense or the sense of whiteness and blackness, I would suggest the word aleucopsia from the Greek, ἀ, priv.; λευκός, white, and hemialeucopsia for the loss of this sense in the visual half fields; for the loss of the sense of form the term amorphopsia, from the Greek ἀ, priv.; μορφή, form, and for this loss in the corresponding half fields, hemiamorphopsia. These terms may be found convenient in description. It is true that when the loss of the light sense is complete that of form and that of color necessarily go with it, but as was seen when discussing relative hemianopsias, we may have incomplete forms of hemialeucopsia.

Hemialeucopsia has received the consideration required in this chapter in the discussion of hemianopsia in its cerebral and especially its cortical relations, and the achromatopsias will be presently considered. A few words might be said rather in the way of suggestion than of detailed consideration about the study of amorphopsia, especially with reference to its bearings upon questions of cerebral localization. Usually the methods of examining the fields for form are of the simplest character, although sufficient for the ordinary purposes of the ophthalmologist. The reader unacquainted with these methods is referred elsewhere and can find them in the works on ophthalmology. An adaptation of the eye to different degrees of illumination is required for a better study of these fields, and particular methods of getting the best results have been described by some writers.

I have already expressed the view that the only primitive cortical centre of vision is that for light or for the recognition of whiteness and blackness, and that color recognition develops ventrally in the expansion of the cerebral surface, the recognition of form probably developing dorsally. In the diagrams, Figs. 5 and 6, the most elementary centres for form are represented as probably situated along the edge of the hemisphere, both on its mesal and lateral aspect; in other words, in the most dorsal part of the cuneus and in the first occipital convolution. Presumably elementary forms are represented cortically in closest relation with the primitive centre for light, the representations for more and more complex forms receding with cortical development from the primitive area of representation.

Little is known about form fields through studies of their extent by means of objects of different shape and character, and it is possible that something of value with regard to cortical localization might be learned by perimetric observations of the fields for form as determined with articles of particular shape or design, instead of confining these investigations to the use of a limited number of geometric figures or to letters in one or two languages. These studies might prove of particular value in cases of partial mind-blindness or of word- or letter-blindness with large retention of vision for other purposes.

CEREBRAL CHROMATIC PHENOMENA AND THE CORTICAL CENTRES FOR COLOR RECOGNITION.

Color-blindness, or achromatopsia, is the inability to recognize colors. It may be total, although this form of achromatopsia is rare, and even when it is present the individual may distinguish as differences in lightness or darkness what others recognize as differences in color. Most frequently the color-blindness is partial, and this partial color-blindness may be of several forms. Hemiachromatopsia is the loss of the ability to recognize colors in one half-field. Central achromatopsia is an affection in which the half visual fields retain the power of color recognition, while this is lost in the central or macular field. Dyschromatopsia is difficulty in distinguishing colors, and parachromatopsia is false or incorrect perception of colors. Partial color-blindness may be regarded from another point of view, as to the color or colors which are lost or disturbed; this is illustrated by such affections as red-blindness, green-blindness, violet-blindness, or red-green blindness. Color amnesia, or color aphasia, sometimes incorrectly spoken of as amnesic color-blindness, is an affection in which the patient, while able to recognize colors by the different tests, is unable to name the colors recognized.

For the sake of a clear understanding of cerebral color affections, it may be worth while to call attention here to a few well-known facts regarding chromatic phenomena. The sensations of color result from dissolving white light into its spectral colors. The so-called primary colors of the spectrum, as green, red, yellow, and blue, each includes somewhat differing sensations of color. These correspond roughly to what are known as shades of color. The power of recognizing these differences varies greatly in different individuals, and even in the same individual at different stages in the course of his education or training. The mixing of certain colors together and the mixing of certain colors with white light produces new sensations of color. A word or two might be said about a few of the terms frequently used in the discussion of color phenomena. Hue depends on the wave lengths of the rays; the intensity on the amount of light falling on a body in the given time, and the saturation on the amount of white light falling on the area in the same time. Mixing of the colors of the spectrum produces white light, or the same can result from the mixture of complementary colors, although in this case the light will differ in intensity from the white light which is the effect of mixing all the colors of the spectrum.

By hue we mean color in the ordinary sense in which the word is used; it is hue which gives red, green, blue, etc. The hue, however, is not always simple and therefore cannot always be defined by making it synonymous with wave lengths. It may be the result of an admixture with various spectral colors, or of white with some colors, and then it can only be expressed by averaging or approximating the wave lengths of the spectral colors. Sometimes the three constants of color are defined as hue, purity or tint and brightness. Purity or tint does not depend upon the wave length, but upon the amount of white light that enters into the colors; the less there is of white light the purer is the color.

Brightness depends upon the energy of the ethereal movement and the sensitiveness of the retina for the particular color. Explained in another way, the brightness is in inverse proportion to the amount of blackness mixed with the color. In the present connection we are only concerned with these general phenomena of color in so far as they have some bearing upon the question of cerebral activity with regard to color sensations. Hue, intensity, tint, and brightness as subjective phenomena all have their cerebral correlatives, but the only constant material substratum is for hue as expressed in fundamental colors.

It has been discovered that in the retina exists one and perhaps several photo-chemical color substances. The best known of these is the visual purple. The existence of this substance has been demonstrated in various ways, as by removing the eyes of a frog after having kept the animal some time in the dark, when the retina is found to be of a red or purplish color, which soon fades, leaving it an opaque white. Investigation has shown that this visual purple is confined to the rods and even to their outer limbs. It is absent from the fovea centralis. It is not present in the eyes of some animals, as, for instance, in the eyes of snakes, whose retinæ are composed almost entirely of cones. It is believed, but not fully demonstrated, as in the case of visual purple, that in the retina are present visual substances for each of the fundamental colors.

As is well known, views differ as to which of the colors of the spectrum are fundamental. Helmholtz believed that they were three in number,—namely, red, green, and violet; while, according to Donders, red, yellow, green, and blue were primary or simple colors. Hering regarded as distinct the recognition of white, black, red, yellow, green, and blue. Other views as to the fundamental colors have been held, but these are usually represented as three or four in number. The hypothesis of three fundamental colors was first put forward by Young. Foster and many physiologists accept the trichromatic view; all agree that while there may be more, it is necessary to recognize at least three primary colors. Each of these probably has its own visual substance, so that in the retina, besides the visual purple, there is probably a visual red, visual green, visual blue, and possibly a visual yellow substance.

The theory is that the decomposition of each visual substance is accompanied by molecular vibrations which are taken up by the retinal structures and transmitted through the various layers of the retina to the optic nerve, and thence by the optic tract, basal centres and optic radiations to the visual cortex. The colors are sifted at the periphery of the visual apparatus and each sifted color has its place of representation in the cerebral cortex.

Young held that the three primary color sensations were represented by three distinct nerve arrangements in the retina and visual apparatus. Whatever view is taken as to the number and character of the fundamental colors, I believe with Young and Helmholtz that in the peripheral end organs are separate structures for their sifting. It is with colors as it is with other forms of sensation. All cutaneous sensations, as, for instance, those for touch, pain, and temperature, undergo a receiving and sifting or differentiating process at the body's periphery. These sifted sensations are carried to the dorsal ganglia, to the spinal cord, to the oblongata, the basal ganglia, and the brain cortex by separate tracts, and it is believed that they have separate centres of representation in the cortex. The phenomena of dissociated cutaneous sensations are best explained on this supposition, and color sensations are dependent upon analogous structures and processes.

Colors are recognized by means of a specially developed apparatus, which, beginning with certain photo-chemical substances in the retina, ends with cell groups located in a part of the cerebral cortex set apart for color recognition. In the retina are differentiated structures for the sifting of different colors, and midway between these and the ultimate cortical centres for colors is a basal relaying station, the pregeniculum, in which colors receive their first central differentiation. Transmitting structures for color sensations, probably also differentiated, are contained in the optic tract and the optic radiations. It is through the workings of this barely sketched complex mechanism that colors are appreciated as separate sensations. The first sifting takes place in the retina, the last in the cortex, and blendings as well as siftings occur in both. These blendings give the different hues and shades of color, those in the cortex being interpreted through cellular associations.

Before taking up the consideration of cortical disease causing color impairment or loss, it will be useful to glance at the various focal affections of the entire visuo-chromatic apparatus capable of producing these affections. Beginning at the extreme periphery, in the first place, one or more of the photo-chemical substances in the retina may be congenitally absent or deficient, or these may be injured or destroyed. Our knowledge is, however, quite limited as to achromatopsias, which have their cause in abnormalities of the color substances; in the second place, disease involving the peripheral nerve apparatus, which takes part in the early stages of color sifting, that is, of the rods and cones and of the ganglia-cells or other cells of the retina, may cause color-blindness or some form of disturbance of color perception. This has been observed, for instance, in some of the forms of retino-chorioiditis involving either the macula or the retinal periphery.

Color sensation may be interfered with or lost by any form of disease or disturbance which impairs or destroys the conducting fibres concerned with the transmission of those retinal stimuli which give rise in consciousness to ideas of color. The optic nerve and tract are subject to such affections as retrobulbar neuritis in toxic amblyopias, and neuritic atrophy in tabes dorsalis. They and the optic radiations may be the seat of embolic or thrombotic lesions causing softening, or somewhere along these transmitting paths may be located hemorrhages, abscesses or tumors. In so far as such lesions interfere with the fibres or fibrils, whose business it is to transmit vibrations concerned with color perception, impairment or loss of the power of recognizing colors will result. Gross or microscopic disease of the pregeniculum, the great relaying station in the visual pathway, will give various affections of the color sense, according to its nature, exact position and extent.

In the occipital cortex are situated separate centres for the recognition of the fundamental colors. Authorities equally high differ with regard to this subject: some, like Holden [20] in a recent paper, arguing that it is not necessary to predicate the existence of cortical color centres in order to explain the normal chromatic phenomena or the disturbances in color recognition which result from disease or injury of the brain; others, like Landolt, Verrey,[21] and Vialet, with whom I agree, believing that distinct centres for luminosity, form, and color are differentiated in contiguous regions of the cerebral surface. Provisionally I would place these centres of chromatic representation ventrally to the primitive centres for light, either in the lingual lobule if the representation of light, that is, of whiteness and blackness, reaches only to the calcarine fissure, or in the fusiform lobule (fourth temporo-occipital convolution) if the primary centre includes, as some suppose, the lingual lobule (fifth temporo-occipital convolution).

It would, of course, be absurd to contend that the almost innumerable shades and tones of color have separate representation in cerebral cortical cell groups. The representation as indicated is probably only for the fundamental colors. What is known as to complementary colors and as to the results of the admixture of different colors in the production of white or gray, and also as to the blending of white or black with the various fundamental colors, suggests the probable explanation of the cerebral processes related to the recognition of the numerous shades and tints of color. Both in the eye and in the brain fine color discrimination is brought about through processes of blending or association; at the periphery through photo-chemical blendings and end-organ selection, in the cortical centres through neuronal and neuro-fibrillary associations.

Wilbrand's [22] hypothesis regarding the centres for color and for light and darkness, already alluded to, is well known and of much interest and needs fuller consideration here. He would have us believe that the cell groups representing color and light are placed in layers or strata, one above the other, in the cerebral cortex of the calcarine fissure. Stratification localization has been favored by not a few neurophysiologists. The representation of the tactile sense, the muscular sense, and of movements have, for instance, been regarded as in superimposed layers in the recognized motor cortex. The methods of the evolution of cerebral structure are opposed to views of this kind. The cells and cell groups which minister to special functions and to association are arranged with considerable uniformity in a similar way in layers and strata in all parts of the brain. As function is added to function and as function is modified in the course of phylogenetic and ontogenetic evolution, the unvarying tendency in anatomical development is towards surface expansion. To accommodate evolving function new regions of the cerebral cortex are added. In this way primary, secondary, and tertiary fissures and gyres have developed. The organ of vision, peripheral and cerebral, at first only capable of appreciating light or lightness and darkness, as evolution advances, acquires the power of recognizing color and form.

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[20] Holden, Archives of Ophthalmology, vol. xxiv. p. 447.

[21] Verrey, Arch. d'Ophthal., par. v. 8, July and August, 1888.

[22] Wilbrand, Ophthalmiatrische Beiträge zur Diagnostik der Gehirnkrankheiten, 83.

According to Wilbrand, the light-dark or white-black centres are located in the deepest layers of the primary visual cortex near the centrum ovale, while the cells representing the different colors are arranged in superimposed strata nearer the cortical surface. In order that sensations of color shall be realized, it is necessary, according to him, that sensations of lightness and darkness shall be first appreciated, and for this reason these perceptive centres are most deeply placed in the cortical cinerea. A visual sensation of light having occurred, this is transferred to superimposed layers. Wilbrand applies Hering's valence theory as to the manner in which atoms combine, to explain what occurs in the process of the recognition of colors. The perception of color is in accordance with the valence relations which the colors bear to lightness and darkness. Red, green, blue, yellow, each of the fundamental colors, has in it its own color valence and besides a certain valence of white light, and in accordance with the destruction or disturbance of this valence will be the degree of impairment of color vision. The perceptive centres for light and colors in the cortex have a power of valence differentiation which corresponds with that possessed by the photo-chemical substances and peripheral nerve structures at the other extremity of the visuo-chromatic cerebral apparatus.

I shall next take up the question of the position of the centres of color representation from the standpoint of clinicopathological evidence. The cases put on record are chiefly illustrations of loss of color recognition in the half-fields; in other words, of hemiachromatopsia. Central or paracentral achromatic scotomata are sometimes observed, and doubtless with closer observation we shall in the future be able to determine central as well as peripheral fields of color recognition with separate cortical centres for both. It is altogether probable also that for color as for light there are lower and higher macular centres, but we are not yet in position to demonstrate this fact.

The clinicopathological evidence as to the position of the centres of color recognition in the cortex is meagre and of an unsatisfactory character. It favors, however, the convolutions situated ventrad of the calcarine fissure as its probable seat. In a case reported by Henschen [23] the lingual and fusiform lobules were involved in the destructive lesion, although, unfortunately for the purposes of argument regarding the color centres, other portions were also implicated.

Two of the most interesting cases of achromatic defect with lesions of the brain demonstrated by necropsy are those of Brill and of Verrey. As the case of Brill,[24] although reported twenty-two years since, has received little or no attention from writers on the subject, I shall refer to it in some detail.

The patient, a man sixty-three years old, in October, 1881, had an apoplectic attack resulting in right hemiparesis, right hemianæsthesia, incontinence of urine, and increased right knee jerk. After several weeks these symptoms were changed and supplemented; the anæsthesia of the thigh became hyperæsthesia, tremor of the paralyzed side developed, and the patient suffered from headache, restlessness and insomnia, with spells of tinnitus and dazing and also dulness of mind. His sight became dull and hazy. Four months after the attack the patient was examined by Dr. Brill and Dr. E. C. Spitzka in consultation. He failed to recognize green; he was doubtful as to whether violet was greenish or bluish; green, he said, was red or rather pinkish; he could always recognize red and yellow. The color loss involved both eyes. Gross objective vision was retained except a subjective blurring; the fields of vision were not limited. The patient was illiterate, so that his condition as to letter-blindness and word-blindness could not be determined. He had worked on the railroad for thirty years before his illness, and was able to recognize all colors.

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[23] Henschen, Klin. u. Anat. Beitr. z. Pathol. des Gehirns, Upsala, 1890, 1903.

[24] Brill, Amer. Jour. of Neurology and Psychiatry, vol. i, N. Y., 1882. Previous to the report of the necropsy, Brill had reported this case clinically in the Chicago Med. Review, April, 1882.

He died about six and a half months after admission to the hospital. At the necropsy a softened area was found at the apex of the left cuneus and the adjoining upper part of the lingual convolution. Brill exactly describes the area of softening as follows: "The focus was triangular, the apex being directed forward and the short side posteriorly. It extended to within a centimetre and a half of the apex of the occipital lobe, and involved half the altitude of the cuneus in the middle of the latter and almost the entire apex of this lobe. The gyrus lingualis was involved for about one-quarter of its width on the surface, and evenly from the posterior almost to the anterior extent of the lesion described (Fig. 14). The calcarine fissure could not be identified excepting its most anterior extremity, and even at this spot both borders were softened. At its lowest border the softening reached almost to the posterior cornu." Brill cites chiefly from Exner's [25] work on cerebral localization a number of cases of impairment or loss of color recognition from cerebral disease.

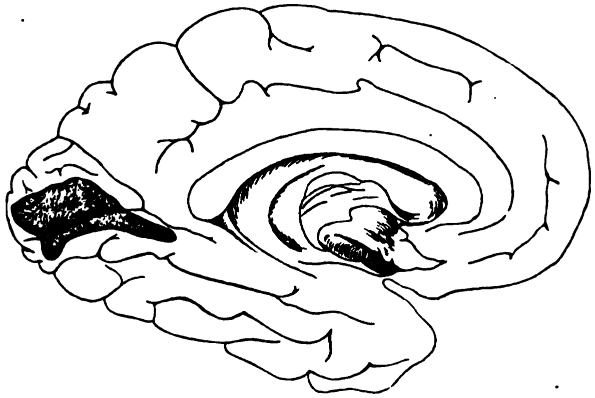

FIG. 14.—Mesal face of the occipital lobe, showing the superficial position and extent of an area of softening causing achromatopsia; the calcarine fissure is indicated where the white interspaces are shown; the lesion involved the inferior portion of the cuneus and a bordering zone of the lingual lobule. (Brill.)

It is unfortunate that Brill in the report of this most interesting case does not clearly indicate whether the color-blindness in both eyes was hemiachromatopsia or an absence of the power of recognizing colors without reference particularly to the half-fields. Nevertheless the case is of value in connection with the necropsy, which clearly indicates that cerebral achromatopsia in this, as in the other cases referred to, was associated with disease of the region ventrad to the calcarine fissure, although it also included this fissure and a part of the cuneus.

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[25] Exner, Untersuchungen über die Localization der Functionen in der Grosshirnrinde des Menschen, Wien, 1881.

Verrey reported, in 1888, the most interesting case of hemiachromatopsia with necropsy, showing the lesion to which the loss of the chromatic sense was due, which has yet been put on record. The patient was a woman sixty years old, whose symptoms were difficulty in reading, with good general visual acuity, slight concentric contraction of the field, complete absolute right hemiachromatopsia, and a sensible diminution of visual acuity for the right half of the visual field. The patient had paroxysmal headache and attacks of vertigo with vomiting. She gradually developed fatigue in reading, with letter dancing and dyslexia. Examination showed that with her central field she could read letters and words written in different colors. Twenty days before her death the patient's symptoms were much the same as above summarized, with the exception of a further enfeeblement of luminous perception. Eight days before her death she fell, and at this time developed a complete left hemiplegia, entire cutaneous irresponse, violent vomiting and clonic and tonic convulsions of the face and right side. The necropsy showed recent large hemorrhage in the right hemisphere destroying the centrum ovale and the basal ganglia, and filling the right lateral ventricle. A fresh hemorrhage the size of a bean was situated at about the junction of the anterior and middle third of the left callosum. A second lesion was found between the floor of the posterior horn of the left lateral ventricle and the basal surface of the occipital lobe. Close examination showed a large thrombosed vessel, and that the parts destroyed were the white substance of the third occipital convolution, more or less completely the white substance of the occipital extremities of the lingual and fusiform convolutions, and the posterior inferior point of the cuneus. The superficial involvement of the lingual and fusiform convolutions is shown in Fig. 15.

The following case of double hemiachromatopsia, recorded by Mackay and Dunlop,[26] also throws some light on the probable position of the cortical color centres. The lesions present were in the temporo-occipital lobe almost entirely ventrad of the calcarine fissure. The patient, a man sixty-two years old, had a sarcoma of the stomach. He had an attack of general disturbance of vision, and examination on the same day showed that he was completely color-blind. Examination showed a double homonymous hemianopsia with greater involvement of the left side. Later the power of vision for light and form improved, while that for color remained permanently lost. Five months after the first attack causing disturbance of vision and color-blindness he had an attack of right hemiplegia from which he died. At the necropsy the optic nerves were found to be intact. Both occipital lobes showed atrophic areas. The atrophy included the posterior parts of the temporo-occipital convolutions on both sides with an extensive destruction of the tissues on the right side. On the left side the atrophy extended deeply and involved a small area of the gray substance of the calcarine fissure. On both sides the lower borders of the optic radiations were affected. The process was supposed to be due to hyaloid degeneration of the vessels. The case was not a pure one of hemiachromatopsia; the visual fields for light and form were to some extent affected.

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[26] Mackay and Dunlop, Scottish Med. and Surgical Journ., December, 1899.

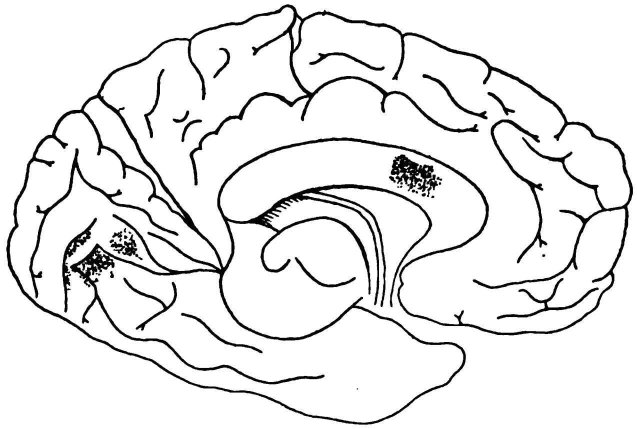

Fig. 15.—Mesal aspect of the left hemicerebrum, showing the cortical extent of a lesion causing hemiachromatopsia; the subcortical radiations of the lesion are described in the text. A small lesion of the callosum is also shown. (Verrey.)

In this case the chief destruction causing the hemiachromatopsia was in the lingual and fusiform lobules (fourth and fifth temporo-occipital convolutions) and the posterior inferior portion of the cuneus and calcarine fissure were implicated. This case again points to the convolutions situated ventrally to the calcarine fissure as the part of the temporo-occipital region most probably concerned with color recognition.

A case recorded by Swanzey [27] carries with it some suggestion as to the site of the centres for color recognition. A man seventy-seven years old suddenly became affected with a slight homonymous defect for light in the left upper quadrant of the field of vision, while over the entire left field color sensation was absent. This case, as pointed out by Gowers, suggests that the centre for colors is adjacent to that part of the half vision centre in which the upper quadrant is represented. It will be remembered that the case of quadrant anopsia of Beevor and Collier seems to show that the upper quadrant of each visual field is represented in the lower part of the cuneus and the calcarine fissure and perhaps in the lingual lobule. If this be true the case of Swanzey would point to the parts adjoining this quadrantic centre, most probably to the lingual or fusiform lobule as the seat of color representation.

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[27] Swanzey, Trans. Oph. Soc., vol. iii. p. 185.

A few other cases of achromatopsia probably due to cortical, or at least to cerebral, disease are worthy of citation, although little is taught by them with regard to the position of the color centres. In a case reported by Eperon,[28] a man seventy years old had suffered with hemiparesis, which almost entirely disappeared except a sensation of numbness in the right arm; suddenly he developed word-blindness. On examination it was found that in the entire right half of the field the sensation for color was missing. Perception of light and acuteness of vision, although present, were considerably reduced. A man fifty-four years old had diminution of vision with glimmerings or flickerings before the eyes for ten or twelve days. He had complete left hemianopsia for all colored objects; he also had hemianopsia for small white objects, but not for those of larger size. His vision was five-sixths of the normal when black characters were on a white ground, but when the characters were pale the vision was reduced to six and a half. Improvement took place, but symmetrical scotomata remained on each side, about fifteen millimetres from the point of fixation. Sensation for colors returned, but not as distinctly as before the attack. After this decided improvement a sudden right-sided hemianæsthesia appeared and vision became worse again. A small defect for white was present in the whole of the upper right quadrant. This case is recorded by Meisling.[29] In another case, which we owe to Ziehl,[30] a man on awakening in the morning found that he was unable to read. Hemianopsia was absent, but right homonymous hemiachromatopsia was present, the dividing line passing vertically through the fixation point. In the affected fields all colors appeared as gray. In a case recorded by Gaudenzi,[31] relative hemialeucopsia and hemiachromatopsia were present.

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[28] Eperon, Archives d'Ophthalmologie, iv. 1884, Juillet-Aout.

[29] Meisling, Ref. Jahresb. f. Ophth., 1897, p. 314.

[30] Ziehl, Verhandl. d. Gesellsch. Deutscher Naturf. u. Aerzte. 67. Vers. zu Lübeck. ii. Teil.; ii. Hälfte, p. 184, 1895.

In a case of hemiachromatopsia, reported by Samelsohn,[32] the autopsy showed a gliosarcoma of the optic tract with some invasion of the thalamus and geniculate body, the optic radiations and cortex being free from disease. In a case of Uhthoff,[33] with normal sharpness of vision, unilateral hemianopsia for color and normal limits for white were present, the autopsy revealing a sarcoma of the pons which compressed downwards the cerebral peduncles and reached into the third ventricle. On the left the tumor had grown into the anterior portion of the median fossa causing protrusion of the dura. The chiasm was much attenuated. The right tract was somewhat contracted in the position of the chiasm. In another case put on record by Samelsohn [34] the patient had some difficulties of vision following an apoplectiform seizure. At this time the entire right side was paralyzed, but when examined by Samelsohn only a slight hemiparesis of the right arm and thigh without any disturbance of sensation could be found. The patient had a typical left-sided hemianopsia for colors. From the vertical dividing line all colors were seen on the right, but none on the left. Bjerrum [35] has reported the case of a man thirty-nine years old, who, after headache lasting fourteen days, developed indistinct vision. The outer limits of the light field were normal, as was also the field for form. Upon examining him with colored objects it was found that he was suffering with complete color-blindness in the left half of the visual fields for both eyes, the dividing line being vertical to the fixation point. All colors in the achromatopsic fields were recognized as gray. The day after he was examined intense headache and inflammatory swelling in the left temporal region were present. An abscess formed; an incision was made, and the patient died, but necropsy was refused.

Besides the case of Mackay and Dunlop, already referred to, a few other cases of double hemiachromatopsia have been recorded. Steffan [36] recites the case of a man sixty-two years old, a printer of colors, who, before the attack, having had a perfect sense for colors, after an apoplectiform attack with dizziness and darkness before the eyes, but without paresis or loss of consciousness, had total color-blindness in both eyes without affecting the sharpness of vision or the visual fields. Examination of the eyes showed nothing abnormal. Examinations were made shortly after the attack and also four years later. At this time he was not totally color-blind, but tests showed pronounced blindness for red and green and diminution for other colors. The central sharpness of sight and the visual fields were still normal. No disturbance was present of the sense of white or black. Later on the sensation for red, yellow, and blue was present, but only when the colors were extremely intense. Alexander[37] reports the case of a man who had, a short time before coming under observation, lost the ability to recognize colors. On examination with samples of colored paper, light green appeared to be yellow, dark gray violet, red, and scarlet and brown, green. The acuity of vision was normal. The eyegrounds were normal, with the exception that they were considerably pigmented. Two years later the condition had changed but little. The color-blindness was extended over almost the entire visual fields. No scotomata were present. A man fifty-four years old, whose history is given by Quaglino,[38] after an apoplectiform attack was found to be completely amaurotic and paralyzed on the left side. The hemiplegia disappeared slowly and the visual power gradually improved. In a year the hemiplegia had disappeared entirely. The visual acuity was now excellent, he being able to read the smallest type and see the small birds at the top of the trees. He had, however, incomplete left lateral homonymous hemianopsia. After the attack he could not recognize any colors but black and white; all faces appeared to him pale and colorless. The color-blindness was not confined to the hemianopsic field, but extended over the fields of both eyes. This patient lost the ability to recall the form and configuration of objects, although he knew what they were when they were first seen by him. In a case of Schoeler and Uhthoff,[39] a man sixty two years old, in full possession of the color sense, developed a right sided hemiachromatopsia. He also had upper quadrantic achromatopsia in the opposite fields. It will be seen that this was not a complete double hemiachromatopsia.

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[31] Gaudenzi, Ref. Jahresb. f. Ophthalm., 1899, 476.

[32] Samelsohn, Ein Fall von Hemiachromatopie. Berliner klin. Wochenschr., 1890, p. 331; Niederrhein. Gesellsch. zu Bonn.

[33] Uhthoff, Berliner klin. Wochenschr., Nr. 12, 1897.

[34] Samelsohn, Centralbl. f. Wissensch., 1881, Nr. 47 u. 50.

[35] Bjerrum, Ref. Centralbl. f. prakt. Augenheilk., 1881, 471.

[36] Steffan, Arch. f. Ophth., xxvii. 1881.

In some cases in which the color sense is affected a person can differentiate between different colors, and yet not have the power to pick out particular colors. He may recognize the fact that red differs from green and green from blue or yellow, or that any one color from one or all other colors, and yet he may not be able to say which is this or that color. In a case of Lissauer[40] the patient could differentiate between colors in wools, but when asked to pick out special colors he made the grossest errors. With his color-blindness a diminution in visual acuity was associated.

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[37] Alexander, Arch. f. Ophth., xx. iii., 102.

[38] Quaglino, Giornale d'oftalmologia, 1867.

[39] Schoeler and Uhthoff, Beiträge zur Pathologie der Sehnerven und der Netzhaut., Berlin, 1884; H. Peters, p. 88.

Under such names as color amnesia and amnesic color-blindness is described a symptom which is exhibited by the inability of the person to name a color which he evidently recognizes. It is a variety of the visual anomia (word-dumbness, optic aphasia) hereafter described, see page 148. The patient may be unable to name any color, or his word-dumbness may be for only one or two colors. In a case recently studied by me, for instance, when a green testing object was presented to the patient, he could not name the color, although indicating that he knew it. Finally he said, " It is the Irishman," showing in this way his appreciation of the fact that it was green. He instantly recognized blue and red; yellow he called a " dirty white." The same answers were given for either the right or the left eye and for both eyes together. When he was given a number of objects of different colors and asked to pick out particular colors, he always did so correctly, thus differing from the patient of Lissauer, above referred to, who could not do this, although he knew that the colors were different. This symptom should, I believe, be termed color aphasia or color anomia rather than color amnesia or amnesic color-blindness. It is probably dependent upon some disturbances of the tracts associating the cortical centres for color with the speech centres, sensory or motor.

Some of the hysterical disorders of the color sense are of particular interest in connection with the discussion of the question of cortical centres for color, both in the lower and in the higher visual fields. The mere fact that these disorders are nearly always, if not always, unilateral, points to unilateral cerebral centres of representation. It is not within the scope of this article to direct attention in detail to this subject, which is considered in chapter xvi. I would like, however, to point out in this connection that the most probable explanation of the phenomena is similar to that which is given for unilateral amblyopia when discussing the crossed amblyopias. The visuo-sensory centres for color are functioning normally, or in condition to so function. The visuo-psychic centres for color are in abeyance or are temporarily functionally disjointed from the primary centres. The affection is psychic, as Bernheim[41] believes. The same explanation holds as would be given for various phenomena of hypnosis.

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[40] Lissauer, Archiv für Psychiatrie und Nervenheilkunde, vol. xxi, 1889–1890.

A few remarkable cases of total uniocular color-blindness have been put on record, the acuity of vision and the fields for light and form being normal. The difficulty of explaining such cases on the doctrine of cortical arrest or destruction is evident. The centres for color, like those for light, are not for each eye, but for the homonymous fields of both eyes; and it is therefore necessary to believe that the centres related to the direct fibres of the optic path on one side and those relating to the crossed fibres on the other side are arrested in development. This, although improbable, is by no means impossible. Congenital arrest may depend upon bilateral defect of centres which work together in function. Uniocular color-blindness is better explained on the theory of deficiency or disease in front of the chiasm, as, for instance, absence of the color substance in one eye, or unilateral defect of the peripheral nerve organs concerned with color selection. In some cases this unilateral color-blindness is partial; it may, for instance, be for red-green or blue-yellow. In bilateral cases of congenital color-blindness, which is the most common form, the powers of visual recognition for light and form are retained, but all colors fail to be recognized. Such a total congenital color-blindness may be due to the arrested development of the entire apparatus concerned with color sensation; or of some one portion of the apparatus, as of its periphery, of its basal centres, or of its cortical centres in their entirety.

AMBLYOPIAS DUE TO DISEASE OF THE VISUAL CORTEX OR OF ITS ENTERING AND CONNECTING TRACTS.

Besides the anopsias, hemianopsias and achromatopsias described as the results of lesions located in certain portions of the areas for vision of the cerebral surface, other forms of impairment of sight, due to disease of the visual cortex, or of its entering or connecting tracts, are sometimes met with and are usually included under the general head of amblyopias. Crossed amblyopia is an affection of sight due to cerebral disease, in which the sole or chief impairment is on the side opposite to the lesion. In the few organic cases of this disease which have been recorded, moderate or slight dimness of vision on the same side as the lesion has been present in connection with the marked contralateral impairment or loss.

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[41] Bernheim: Br [illegible] 1893

The discussion of the subject of crossed amblyopia is intimately related to that of cortical macular representation. I have already indicated my belief in the existence of an area of macular representation in the primary or lower visual centres, and I have subscribed for the present to the view of Henschen that this macular centre is probably situated in and around the anterior extremity of the calcarine fissure. In the higher visual zone, in the angular or angulo-occipital region, is also situated a cortical area for the macula. The lower macular area subserves clear vision in what might be termed its most primitive form. Luminosity, or, rather, whiteness and blackness, are here perceived; objects here first receive their clearest recognition. The centre, however, is purely perceptive; it is simply a part of the visuo-sensory area of Campbell and Mott. Natural objects, faces, persons, designs, words, letters or numbers are perceived through the functioning of this lower macular cortical area, but without a recognition of their significance; they are perceived, but they are not apperceived. Perception by the lower macular centre does not evoke concrete ideas the result of visual memories. Such visual ideas are the outcome of the activities of the higher visual field, including its macular subdivision.

It may be well to direct attention here to the different views which have been held with regard to macular representation. While Henschen, whom the writer had chosen to follow, would limit it to the anterior and ventral portion of the cuneo-calcarine visual centre, others have suggested as its location the posterior extremity of the calcarine fissure and its bordering cortex. Von Monakow holds that the macula has a variable representation spread out over both the lateral and mesal aspect of the occipital lobe; in other words, over both the lower and higher visual fields. The facts at our command do not seem to me to support this view. The physiology of sight and clinical experience point to a more or less concentrated cortical representation of the macula.

From his own experiments and those of Schäfer and Munk, Ferrier argues that the angular gyres are more particularly related to the areas of distinct vision,—that is, that they represent the macula. He also holds that the macula is represented in both hemispheres, but most in the angular convolution of the opposite side. His views are indicated schematically in Fig. 16.

Lannegrace[42] and Ferrier and Yeo, who have performed numerous extirpation experiments on the occipital lobe and the angular gyre of the brains of monkeys, hold that destruction of the occipital lobe causes no appreciable impairment of vision, while destruction of the angular convolution causes crossed amblyopia of a temporary character. In one of Ferrier's most striking experiments he removed one of the eyes of a monkey and then destroyed the angular convolution of the same side; this caused total blindness in the remaining eye of the opposite side, as was completely verified by numerous experiments. The animal, however, showed some signs of returning vision the next day, but died as the result of exhaustion, the weather being extremely cold. In another experiment Ferrier carefully examined the condition of vision in a monkey in which he had completely destroyed both angular gyres, and recorded the results as follows:

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[42] Lannegrace, Influence des Lésions Corticales sur la Vue, Archives de Médécine Expérimentale, 1889.

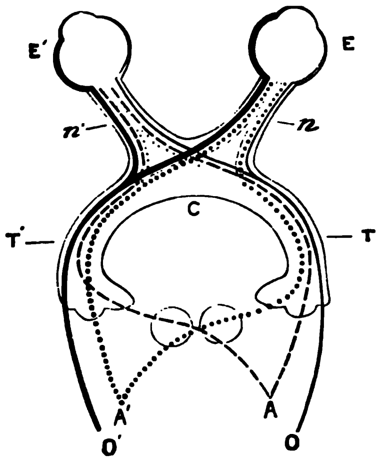

Fig. 16.- Scheme of the optic tracts and visual centres. A, the right and A', the left angular gyrus; C, optic chiasm: E, the right and E', the left eye; *n*, the right and *n'*, the left optic nerve; O, the right and O', the left occipital lobe; T, the right and T', the left optic tract; the thin continuous line represents the retinal relations of O; the thick continuous line represents the retinal relations of O'; the interrupted line indicates the retinal relations of A, and the dotted line the retinal relations of A'; the relations of A and A' with the eye on the same side are indicated by finer interrupted and dotted lines respectively. (Ferrier.)

"There was no ptosis, the ocular movements were normal, the conjunctival reflexes unimpaired, sensibility was intact everywhere, and the motor powers were perfect, but for four days at least the animal was evidently absolutely blind. When urged to move it ran against every obstacle in its path, paid no attention to threats, could not find its food, except by groping, and appeared insensible to light flashed in its eyes. On the fifth day there were evidences of returning vision. It did not knock its head against obstacles; would not walk over the edge of the table; showed signs of perception of light flashed in its eyes and occasionally seemed to wince when threatened. Vision gradually improved, but continued very imperfect, especially for minute objects which it rarely, if ever, seized quite precisely; groping at them with the whole hand, and reaching short, or over, or to the side. It appeared to see objects held above, below, or to either side much better than those held in front of its eyes. Six weeks after the operation my colleague, Professor McHardy, examined the animal, which was very docile with me, testing every portion of the visual field by pieces of apple suspended by a delicate thread. It was concluded that vision was better in every part of the periphery than in the centre. Objects held directly before the eyes and at a little distance were apparently not clearly seen, and never laid hold of with precision. The condition remained unchanged for three months after the operation, similar tests being from time to time applied, and with the same result. I noted also that the animal, when examining any object, always held it at full arm's length from its eyes. The phenomena observable in the animal were such as would be best explained by impairment or loss of central vision."

A few cases have been recorded by Demange, Sharkey, and others which appear to indicate that a destructive lesion of the angular gyre of one side in man will cause loss or great impairment of vision of the opposite eye, with contraction of the visual field of the same side. In a case recorded by Demange,[43] complete loss of sight on the side opposite to the lesion was present, as well as hemianæsthesia and loss of hearing, taste, and smell. The lesion in this case was a large subcortical area of softening extending beneath the convexity of a large part of the right hemisphere. The mesal aspect of the hemicerebrum and the internal capsule were nowhere implicated.

One of the cases frequently referred to and especially used by Gowers and Ferrier in support of the view that crossed amblyopia is due to lesion of the angular convolution, is that of Sharkey.[44] In this case there were left hemiplegia, more marked in the arm and leg than in the face, dimness of vision, impairment of hearing on the left, loss of taste and of common sensibility on the left half of the tongue, and also loss of sensibility in the entire left half of the body. The paralysis of the leg largely, and the anæsthesia entirely, disappeared. The entire right hemisphere was much reduced in size, and, according to the report, the convolutions supplied by the right Sylvian artery were nearly all necrosed and atrophied. The crossed amblyopia was one of the most interesting of the clinical phenomena present. The blindness in the left eye was almost complete at first. Improvement in sight, as in the other special senses, took place in about four weeks, and in six weeks and a half vision was completely restored.

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[43] Demange, Revue De Méd., May, 1883, p. 391

[44] Sharkey, Med.-Chir. Trans., London, 1884, p. 265

A number of clinical cases without necropsy, in which crossed amblyopia has been present, have been recorded by Ferrier and others. In these cases a series of other unilateral phenomena have, as a rule, been associated with the dimness of sight, these being usually such as hemianopsia, hemianæsthesia, partial or complete impairment of other special senses, astereognosis, and hemiparesis. Doubtless in not a few instances the partial amblyopia is present in organic cases with unilateral symptoms, but is overlooked. In one of the cases reported by Ferrier, the patient was unaware of the impairment of vision in the eye on the side of her other symptoms until her attention was called to the fact by special examination. Not infrequently one eye with perfect or good vision serves so well the purposes of the individual that impairment of sight in the other does not attract attention until some incident or investigation reveals its existence. In these clinical cases of crossed amblyopia, marked impairment of the color sense is often present. In one case green was the only color recognized. The visual fields, determined with such sight as is left, are greatly contracted.

Gowers gives brief notes of a case which is instructive in connection with this question of crossed amblyopia. The patient was a woman fifty years old whose right eye had been removed many years before. She had marked concentric restriction of the field of the left eye accompanied by amblyopia. Accompanying symptoms were left hemianæsthesia, dulness of the senses other than sight, and persistent headache. The patient was not an hysteric, but syphilis was probable. The patient's amblyopia and other symptoms improved under the use of iodide.

While these cases undoubtedly show the possibility of the occurrence of a form of crossed amblyopia from lesion of the angulo-occipital region, it is not to be inferred that they prove that this region is the only area of representation of clear or macular vision. In the cases of Demange and of Sharkey, the lesions were such as to involve the angulo-occipital subcortex, and may have severed the connections between a lower and higher macular centre or even the macular bundle from the pregeniculum to the primary macular centre. The now somewhat numerous cases of precortical and cortical word-blindness, with or without letter blindness, have not shown, or at least not in the records given, that clear visual perception was lost. Such cases sometimes have normal acuity of vision for objects other than words and letters both near and remote, as in one case studied by me recently. If the angulo-occipital cortex is the sole area of clear or macular vision in man, acuity of vision for all purposes would suffer in such cases, instead of the defect being confined to vision for words and letters. The angular gyre is simply a part of the higher visual or psycho-visual area, the particular part in which is located vision for the highest and last form of visual symbolization—that of language.

The experiments of Ferrier and Yeo and Lannegrace might be interpreted as indicating that even in the monkeys experimented upon there is a higher area of clear or macular vision located in the position asserted by Ferrier. These animals, from destruction of the angular convolution on the one side, had transient loss of vision in the opposite eye, and even when the same region of the opposite side was destroyed, the total loss of vision which resulted was partially recovered from if the animal survived sufficiently long. This does not necessarily show that the cortical area destroyed was the only area for clear or macular vision, but, taken together, the experiments might be regarded as showing that lower as well as higher macular cortical centres are present in the monkey as well as in man. In the man these centres, as the result of evolution, become cortical areas of representation of designs and symbols; in the monkey they are probably the seat of memorial images of objects of small size and particular form. The fact must also not be lost sight of that in the experiments upon the monkey, the macular bundles, geniculo-cortical and intercortical, or both, must have been injured or severed. I believe in an angulo-occipital macular area in the human being, but it is one which is concerned not only with clear or macular vision, but with vision whose purpose is the recognition of words, letters and numbers, and probably also geometric and other designs. The proper place for the further discussion of the symptoms which result from destruction of this higher macular area of the cortex is where word-blindness and other forms of visual aphasia are later considered.

It is not improbable that there is for the macular field, as for the peripheral fields, a half representation, both primary or lower and secondary or higher, in each hemisphere; in other words, that a strictly limited lesion of the calcarine cortex on the one hand and of the angular region on the other may cause blindness in half of the macular field of the corresponding sides. At the same time it is probable that this half macular representation is not so strictly defined by a vertical dividing line as is the peripheral retinal representation. Putting it in another way, the macular centre of one side is more or less representative of the macular fields of both sides.

Elsewhere [45] I have recorded two interesting cases which seem to indicate that a very limited lesion of the cortical area for word-vision or of the tract leading to it from the lower macular centre, might cause a loss of vision for one-half of a word, what might be termed hemianopsic amblyopia. One-half of the central field is obscured or lost, the peripheral field being maintained, or it may be partially obscured. The patient is half blind for words. A word of more than four or five letters will only be half seen, or at least will be partially cut off, unless the patient turns his eyes so that the unimpaired half of the central field can take in different parts of the word in succession.

It is probable not only that lower and higher cortical macular centres exist, but also lower and higher peripheral representation in the cortex; it is probable, moreover, that the lower macular region is connected with the higher macular region by its own bundle, and the lower peripheral portion of the fields with the non-macular portion of the higher visual field by its own fibre tracts. It follows that certain symptoms will result from disease of the lower macular centre or of the tracts going from it to the higher centre of the same side and to that of the other side; it also follows that certain affections will result from lesion of the fibres connecting the lower and higher peripheral cortical visual fields. The macula is not only the organ of acute vision, but of the highest form of psychic vision. The retinal periphery and its lower and higher cortical areas of representation are concerned with perceptions and apperceptions of a lower order.

With the facts just given before us, in order to apply the views advanced to all clinical macular phenomena, let us glance for a moment at the different forms of focal disease, peripheral and central, giving macular symptoms. Macular or central amblyopia will of course be produced by the destruction of the macula itself, but lesions of intraocular origin come almost exclusively under the attention of the ophthalmologist. I would, however, direct attention to the fact that the macula is the part of the retina which is chiefly concerned with the recognition of words, letters and numbers. Complete destruction of a large portion of the macula produces what might be termed, for convenience, a peripheral alexia and visual agraphia, while at the same time very considerable peripheral vision for form remains; in other words, the patient suffering from a large degree of macular destruction will still be able to orient himself with his retinal periphery and also recognize natural objects, individuals, buildings, and, to a considerable extent, designs and structures of various sorts.

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[45] Mills, The Nervous System and its Diseases.—Diseases of the Brain and Cranial Nerves, p. 760. J. B. Lippincott Company, Philadelphia.

Central amblyopia results from a small infiltrating lesion, a nodule of sclerosis or a focus of hemorrhage, for instance, which may by rare chance be so situated as to destroy the macular bundle alone in the optic nerve and tracts in any part of its course. Clinical experience has shown that this bundle is especially liable to be affected by certain toxic agents.

As the fibres of the macular bundle are undoubtedly distributed to the pregeniculum, complete destruction of this body or of a special portion of it, should cause central amblyopia. This central amblyopia would be of the crossed variety, and if only one external geniculate body were destroyed it would be transient, or would at least be recovered from in part. It is not yet positively proved that the pregeniculum is the basal centre for the macular bundle alone, as is held by Knies[46] and others. Some facts point otherwise, as for instance the case reported by Henschen, which seemed to show that hemianopsia in the lower quadrant may be caused by a lesion of the dorsal part of the pregeniculum. In any case, if the periphery of the retina is represented in the pregeniculum the macula is also represented in this ganglion, and if the terminus of the macular bundle is involved in a completely destructive lesion, crossed amblyopia, as above indicated, would result. Partial lesions of the basal macular centre, on one side or both, cause scotomata which may be transient or disregarded if the lesion is unilateral, or persist in consciousness if bilateral geniculate lesions are present. Paracentral scotomata, peripheral vision, and central vision in the main remaining intact, should result from lesions which only partially destroy the macular geniculate centre. Such a clinical case has been recorded by Knies. In this case diminution of the hemianopsic pupillary response was present, that is, light thrown on one side of the retina caused a prompt iritic response, while on the other side the reflex was sluggish. A form of dyslexia apparently due to the impairment of macular vision was present in this case, which would seem to bear out the view that the macula is the part of the retina chiefly concerned with word- and letter-vision.

Leaving the basal centres, a lesion of the macular bundle in the optic radiations should cause some degree of central amblyopia; this would be transient and more or less partial according to the degree of involvement of the fibres constituting this bundle. Here, as in the case of the pregeniculum and the optic tract, small destructive lesions would be represented symptomatically by scotomata. Bilateral lesions of the occipital macular bundle would cause permanent amblyopia. In all the cases which have been considered, the amblyopia would be of the crossed variety from unilateral lesion, and in all probability close examination would show some impairment of vision with contraction of the field on the side of the lesion.

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[46] Knies, The Relation of the Eye and its Diseases to the Diseases of the Body, New York, 1894.

In our consideration of this subject we have now reached the macular centre in the primary or lower cortical visual field. Presuming the existence of this centre, have we any clinical evidence in support of this view? As has already been stated, its existence is predicated by the occurrence of hemianopsia less a small central portion of the half field near the fixation point from a cuneo-calcarine lesion, and it is made still more certain by those cases of double hemianopsia in which a small central area of acute vision remains. Cases which suggest a lower macular representation are those in which outer portions of the retinal half field retain vision, while the rest of the half field to the dividing line is obscured. Here again also the question of paracentral and central scotomata becomes of interest. A lesion partially destructive of the cortical macular centre of one side should cause a scotoma, paracentral or central according to its position. This would doubtless be transient or disregarded, but might be elicited by special investigation. The cortical macular centre of the opposite side would serve the purposes of vision, especially if the nasal macular centres were intact. Bilateral disease of the cortical centres for the macula causes persistent scotomata, or central amblyopia if the lesions completely destroy the centres on both sides. Higher central vision, that for letters and words, for instance, would necessarily go with the partial or complete destruction of the primary visual centres, although, as has been indicated, the reverse is not the case. Word-blindness or letter-blindness may be present without destruction of central visual acuity for other purposes than the recognition of language symbols, but a lesion or lesions destroying primary central perception would necessarily disturb or destroy central apperception.

The manner in which hemianopsia from cortical or cortico-subcortical lesion sometimes clears up or rather diminishes, indicates how a central or paracentral scotoma might be produced from a lesion limited to the lower cortical macular centre of one side. Hemianopsia in some cases diminishes with comparative uniformity from centre towards periphery, the region of visual perception gradually expanding. In other cases it may clear in a lower or upper quadrant, or in an irregular area in any portion of the hemianopsic field. In still others, however, an irregular scotoma may remain as the field clears peripherally. In a case of this kind a portion of the cortical macular field has been completely destroyed, the same being true of that portion of the peripheral cortical area farthest removed from the macular centre. An intervening area of both central and peripheral representation has escaped complete destruction.

As already indicated, I believe that a distinct bundle passes from both primary cortical macular centres to the higher centre for clear vision on each side. The tract from the lower centre of the same side probably passes by way of the occipito-parietal subcortex, while the tract from the other side crosses through the splenium of the callosum to conjoin with it, the conjoined tracts going to the angular convolution. A lesion of this conjoined tract or lesions of the tracts from both primary centres before they unite would, of course, cause disturbance or loss of the functions of the higher centre for which they are destined ; in other words, as will be shown more fully later, if the precortical lesions were left-sided, a variety of word-blindness would occur. Doubtless also close examination in such cases would show, at least at first, some impairment of vision in the eye of the side opposite to the lesion, this being quickly recovered from because both eyes are represented in each macular centre.

Recently, in conjunction with Dr. C. D. Camp [47] I have published the history of a case of unusual interest in connection with the subject of crossed amblyopia, and also of that of visual hallucinations the result of organic disease of the cerebral cortex and subcortex. The patient, a woman sixty-three years old, had suffered with impairment of vision in both eyes for nearly two years before coming under observation. This dimness of vision was attributed to glaucoma for which a double iridectomy was performed some months before coming under observation. After this operation her vision was fairly good. I am indebted to Dr. G. C. Harlan, who performed the operation for the relief of the glaucoma, for notes regarding the state of the patient's vision and eyes at this time. Sight was reduced to 20/40 in the right eye and 20/50 in the left and the disc of each eye was cupped to the extent of about 1.50 D. The fields were practically normal with the exception of a slight peripheral cut in the right eye.

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[47] Mills and Camp, Trans. of the American Medico-Psychological Association, vol. xii. 1905; also Amer. Jour. of Insanity, 1905.

I saw this patient first in September, 1894. A few days before my visit she had a cerebral seizure in which the right eye became totally blind and vision in the left eye was greatly impaired. She had also lost the power of standing and was abnormally excitable.

She was brought to Philadelphia where she was attended until her death by Dr. Harlan and the writer. A careful examination of her eyes by Dr. Harlan showed that in the right eye she had scarcely more than light perception. The cupped nerve had filled up to the level of the retina. In the left eye vision was reduced to [illegible] and the field was peripherally contracted to [illegible] degrees on the nasal and to [illegible] degrees on the temporal side. The disc was cupped but the fundus was otherwise normal. Later this patient developed acute delirious mania with vivid and varying hallucinations of sight, which are described in some detail in the paper to which reference has been made.

The patient died about three months after her sudden attack of loss of vision.

Macroscopic examination showed the arteries at the base of the brain highly sclerotic and both pregeniculæ atrophied.

In sections from the calcarine fissure from each side were seen areas of intense congestion, with the formation of numerous very fine new capillaries. These were mostly in the second and third layers of the cortex, and the pyramidal cells in their vicinity appeared degenerated on examination by the Nissl method. Sections from the cortex of the lateral surface of each occipital lobe, near the occipital pole, showed areas [illegible] of a similar vascular appearance, as in [illegible] a part of the same pathological process. [illegible] of these areas were of the pyramidal [illegible] their normal shape and structure completely. The [illegible] beneath these areas appeared degenerated by the Weigert method [illegible] and full of small holes as though nerve fibres had [illegible]

The left optic nerve [illegible] one-third of the normal size of an optic nerve, and [illegible] a greatly thickened sheath. There was much connective [illegible] between the nerve bundles, which were small and [illegible] appeared to be degenerated by the Weigert [illegible] there was no round cell infiltration about it.

Whether [illegible] one eye and the serious loss of vision in the [illegible] constituting a genuine case of crossed [illegible] reported by Ferrier and Gowers, and [illegible] region or of the macular bundle, may perhaps be regarded as doubtful. If due to the lesions undoubtedly present in the calcarine cortex, this crossed amblyopia is of much interest in connection with the question of the existence of a separate macular representation in the primary or lower cortical visual centres of the calcarine region and in the higher angulo-occipital visual areas. Dr. Harlan agreed with me that the final amblyopia in this case could not be attributed to the preceding glaucoma, at least not to the peripheral conditions which were present. With the exception of the cupped nerves, the fundus of each eye was normal and the filling up of the nerve cup seemed to indicate the occurrence of some intracranial lesion. The attacks of sudden loss of vision which occur in chronic glaucoma may, in some instances, be due to intracranial lesions affecting the cerebral, and especially the macular, bundles or centres. It is possible of course that the atrophy of the left optic nerve and tract may have accounted for the great loss of acuity of vision and contraction of the visual field in the left eye, but in any case the sudden amblyopia in the right eye was doubtless due to a sudden vascular lesion in the left hemisphere.

The phenomena of hysterical unilateral amblyopia would seem, when considered carefully, to confirm the theories held regarding crossed amblyopia as the result of a unilateral organic lesion. In unilateral amblyopia of hysterical origin, which is usually but not invariably associated with unilateral impairment of other senses, as of touch, pain, temperature, hearing, smell and taste, the vision is greatly impaired on the affected side. Examination shows in addition concentric contraction of the field on this side. At the same time, some dimness of vision and less contraction of the field may be present on the other side. The same is true with regard to color phenomena. Dyschromatopsia may be marked on the affected side, and affected to a less and perhaps a slight degree on the other. These phenomena are the counterpart of what has occurred in the recorded cases of crossed amblyopia from organic lesion, the differences, if any, being in degree as to the impairment of vision, and also in the fact that the hysterical affection is usually transient, while the other is permanent or at least much more persistent.

Whatever theory may be accepted in explanation of the hysterical amblyopia, it is clear that the affection is in some way dependent upon interference with the functions of the visual centres of one hemicerebrum. Whether this is in the nature of an angiospasm of cortical vessels, as once held, or of a temporary impairment of nutrition of the cortical cells, the fact remains that the symptoms point to a unilateral cerebral disturbance. It is significant that in hysterical cases hemianopsia in any of its varieties is not present, but that the loss of vision is central or general for the side affected. This is similar to what occurs in the experimental lesions produced by destruction experiments upon the monkey, or in cases like those of Sharkey, Demange and others, in which in the human subject cortical or subcortical substance has been destroyed. All the cases, organic or functional in man and organic in the lower animals, show the possibility of the occurrence of a crossed affection of vision distinct from that which gives anopsias or hemianopsias for light, color and form. It is true that the recorded cases of crossed amblyopia are few in number, but as Ferrier suggests, and as one or two of his clinical cases seem to demonstrate, the amblyopia in some instances may be overlooked. Whenever a patient presents such symptoms as hemianæsthesia and astereognosis, the case should be examined for unilateral disturbance of vision and of the other senses. The investigator should not be satisfied with demonstrating that hemianopsia is not present, or even that some central vision is retained in both eyes, but should examine not only for the presence of amblyopia, but for the degree of the retention or loss of central vision in both eyes.

The visual auræ of epilepsy, like the spectra of migraine, have been considered clinically in the present volume by Dr. Lloyd. I shall only direct brief attention to the well-known facts which indicate that these auræ and spectra are usually the evidences of cortical disease, indicating either discharge or inhibition or both of the cortical centres for sight, primary and secondary. These auræ take the form of either simple sensations of luminosity, of colors, or of persons or objects or of groupings of these.

In many instances the primary visual phenomenon is a light sensation, as a flash or a ball of fire. In other cases the aura is a sensation of color which often assumes some definite form, as that of a ball or a star. It is significant that the colors seen are those which are recognized by most authorities as primary colors, as red, blue, green or yellow, or, in rare cases, purple or violet. In the recorded cases red and blue have been most frequently present, sometimes alone, sometimes in combination. One or the other has been almost invariably present. Auræ representing visual ideas are comparatively infrequent, but have nevertheless been observed in considerable number. These in particular cases have taken the appearance of an individual in a certain colored dress, as for instance, a little old woman in brown. The primary sensation of light or the sensation of color in a few cases, has preceded in the same seizure that of some objects representing a visual idea; and Hughlings Jackson has explained this sequence of events by the discharge of primary visual centres followed by that of secondary or higher visual centres. The latter, however, may take place alone.

Some of the most carefully recorded observations regarding the spectra of migraine show that these, whatever form they may take, tend to occupy only the peripheral portion of the retinal field. The central field is nearly always free from these spectra, and the zigzag or other lines diminish in length as they approach it; but this central field is subject on the other hand to inhibition, becoming obscured or absolutely darkened.

It is only necessary in this connection to emphasize that the clinical phenomena thus recorded clearly indicate in the vast majority of cases that the cause of the phenomena is the discharge of centres and areas of the cortex which either act independently or in more or less frequent association. Inhibition of cortical visual centres is indicated by the diminution or loss of sight which occurs as the sequence of luminous and chromatic sensations.

THE VISUAL PORTION OF THE CEREBRAL ZONE OF SPEECH AND VISUAL APHASIAS.

Under the general designation *visual aphasia* are included all those forms of interference with speech and with writing which result from lesion or disturbance of the visual centres concerned with language and the tract associating these centres with lower, co-ordinate, and higher cerebral centres and areas. While our concern is chiefly with the visual aphasias and the visual subdivision of the general zone of language in the cerebrum, a glance at this zone in its entirety forms a proper introduction to the discussion of those parts of it which relate to speech as it is understood through sight.[48]

The cerebral zone of speech includes an extensive cortical region, probably not less than one-third of the cortical expanse of the lateral aspect of the left hemicerebrum, as shown in the shaded portion of Fig. 17. While for descriptive purposes it is best to designate it as a cortical area, it must be understood that both the cortex and the sub-cortex of the entire region enter into its formation.

Within this great zone are several important centres or sub-areas concerned with particular attributes of language. In Fig. 17 these centres are WH, a centre for word-hearing, the auditory portion of the zone of language; at S and S a great centre for speech, often called motor; at WS, LS, NS, a visual subarea concerned with the representation of words, letters and numbers; at W a graphic or writing centre, usually spoken of as the motorgraphic centre; and at N a naming centre in which it is believed by those who hold to its existence that names are correlated with the concrete concepts of the objects named.

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[48] A sketch of the cerebral zone of speech given in the text and the diagram illustrating it are taken from the article by me published in the American Journal of the Medical Sciences, September, 1904, the text of this article having been adapted to the present chapter.

Briefly stated, the auditive centre for speech is situated at the junction of the first and second temporal gyres about opposite the point where the horizontal branch of the Sylvian fissure turns upward posteriorly; the dynamic or motor speech centre occupies the hinder portion of the subfrontal or third frontal convolution, and, according to the view of the writer, the insula or at least its anterior half; the visual centres immediately concerned with speech are located in the angular gyre; the site of the graphic or writing centre is the caudal third of the second frontal convolution, and the naming centre is probably situated in the third temporal convolution.

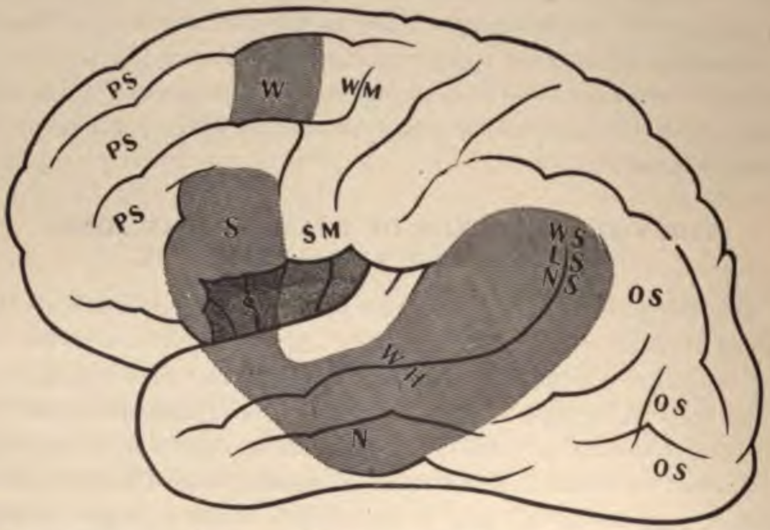

FIG. 17.—Cerebral zone of language. *W*, writing centre; *S*, *S*, speech centre (language arrangement) for emission; *WH*, word-hearing or auditory centre; *WS*, word-seeing; *LS*, letter-seeing; *NS*, number-seeing; *N*, naming-centre; *PS*, *PS*, *PS*, higher psychic region; *WM*, centre for movements of upper extremity concerned in writing; *SM*, centre for movements concerned in speaking; *OS*, *OS*, *OS*, area of object-seeing.

Connecting these centres with each other, and also with other portions of the brain, are subcortical tracts composed of the processes of the cell bodies which are located in the cortex and of their fibrillary constituents. These associating tracts are of a complicated kind, although their number and arrangement are capable of analysis if sufficient time and study are given to the matter. Only one or two points regarding them need here be emphasized,—namely, that the different centres are connected each with the other by a system of to-and-fro association, so that the impulses from special areas probably pass in both directions.

Surrounding the zone of language on all sides are centres and areas which have important relations with the function of speech. These are of at least three kinds, and an understanding of them may be reached by a study of Fig. 17. On the sensory or receptive side of the brain are areas or centres concerned with such functions as object-seeing and object-hearing. Objects which are not recognized cannot of course be named, and these centres for object recognition therefore are necessarily contributors to the sensory concept side of the zone of language. They are represented in Fig. 17 at the points marked OS. On the motor or emissive side of the cerebral speech zone are centres at SM and WM which are concerned with the execution of the work prepared for them by the speech and writing centres. They are, in other words, the centres of representation of movements of articulation and enunciation, and of the movements of the hand and upper extremity generally. At PS in the prefrontal portion of the brain is a region where the highest psychical functions are especially represented. It is evident that in using language, attention, volition, and reasoning play a part, although this part may vary within wide limits. This higher psychical region, therefore, has its particular relation to the zone of language, and especially to its dynamic side.

Let us now concentrate our attention on that subdivision of the cerebral zone of speech which is especially concerned with visual language, its lesions causing the different varieties of visual aphasia. As indicated in what has just been said, at WS, LS, and NS in Fig. 17 is situated the cortical centre for one of the forms of higher visualization, the centre or region in which are stored the visual images of words, letters and numbers. This portion of the cortex is, as has already been shown, one of the subdivisions of the concrete memory field. It is connected on the one hand with the lower or primary cortical areas for vision in both hemispheres; and on the other, with the areas for speech, writing and movements in the cortical zone cephalad of the central fissure, while at the same time it has numerous associating tracts with centres—contributory or co-ordinate in function—like those for object-seeing, for stereognostic perception, for hearing, taste and smell. It is also connected with the special centre or area (N), whose chief function is the association of names with objects, concepts of which are obtained through the special senses. If the reader has a clear idea of the location and connections of this higher portion of the visual area, which is usually spoken of as the area for word-seeing, he can readily comprehend the different varieties of aphasia which result when this centre, its outlets or inlets, or its associated or associating structures are diseased.

The most important varieties of visual aphasia and allied disorders are: (1) mind- or object-blindness; (2) word-blindness; (3) letter-blindness; (4) number-blindness; (5) alexia, paralexia and dyslexia; (6) visual agraphia; paragraphia and dysgraphia; (7) optic aphasia or word-dumbness. Some objection might be made to including mind-blindness in the category of visual aphasias, and such objection is doubtless valid, but as a matter of convenience and with some scientific basis, I have thought it well to include mind-blindness under the general head of visual aphasias. The patient who is unable through sight to recognize a familiar person or object, or to recall the visual memories of such person or object, cannot of course name that of which he is not cognizant, and hence is aphasic in this indirect sense. Psychic blindness is, however, a disorder distinct in most respects from visual aphasia, and it will be therefore understood that it is only included here because of convenience, and because it is so frequently associated with the true cerebral sensory disorders of speech.

MIND-BLINDNESS.

Mind-blindness is that symptom or condition in which the individual, because of cerebral lesion or disturbance, is unable to recognize by sight once familiar objects. Old visual memories of the kind required for recognition of particular objects are no longer recalled.

In order that mind-blindness shall result, it is necessary that the disease shall be situated either in the cerebral cortex or in the tracts which associate certain parts of the cortex. Such disease being present, all other parts of the intracranial apparatus may be normal, and yet the patient will exhibit this symptom.

Numerous synonyms of mind-blindness are used and serve to a certain extent to confuse the subject. The most important of these are soul-blindness, psychic blindness, and object-blindness. Visual apraxia, to which brief reference will presently be made, is still another synonym, and Wyllie has suggested the term visual pragmatagnosia, from the Greek *πρᾶγμα*, an object; and *ἀγνωσία*, a not knowing. It is possible to have lost the power of recognizing an object at sight and yet be able to recall the appearance of the object; or both the ability to recognize the object at sight and to recall its image may be lost together. Wyllie has suggested that the term visual pragmatamnesia be applied to the loss of the power of calling up in memory the appearance of an object. A little consideration will show that in the difference between visual pragmatagnosia and visual pragmatamnesia we have a distinction similar to that which is drawn between cortical word-blindness and pure word-blindness, or between true color-blindness from cerebral disease and cerebral color amnesia. When the patient has lost the power of recognizing an object by sight, but retains the ability of recalling it as a visual memory, the cortex concerned with the function has escaped disease, but the tracts associating it with the lower or primary centres for vision are destroyed; just as in cortical word-blindness the visual memories of words cannot be recalled, whereas in the precortical, or so-called pure word-blindness, the visual memories can be recalled, but the individual is not able to recognize words through vision.

The term mind-blindness or object-blindness might in a general sense be made to cover word-blindness, letter-blindness and number-blindness; indeed these are but varieties of a general object-blindness. As our knowledge of cerebral function and cerebral disease increases, it is probable that we shall be able to classify mind-blindness into a number of sub-types. The uncivilized, like the higher forms of lower animal life, have keen powers of recognizing natural objects and the living beings around them. As evolution advances, added powers of recognition are developed. Doubtless some of these, like the ability to recognize colors with their various shades and tones, artificial designs and geometric forms have their special regions of representation in the general area of object seeing; and disease limited to such special areas or centres gives rise to special types of mind-blindness. The area concerned with the recognition of words and letters is not the only one developed in the educated or trained human being, but it and another sub-area of large extent concerned with the recognition of persons, places and objects, natural and artificial, are all that have so far been differentiated. It is for the reasons here indicated that I have subdivided the area for object seeing as shown in Fig. 3 and Fig. 5. While mind-blindness in its known forms is often associated with word- and letter-blindness and other forms of visual impairment or loss, this is not always the case; and in a few recorded instances the inability to recognize faces or persons has been greater than that for objects of various sorts. A woman in advanced years under my care, who as the result of one or more cerebral seizures, suffered from word-blindness, mind-blindness and other often associated phenomena, was entirely unable to recognize her own sons by sight, although she was largely able to recognize her surroundings and orient herself with regard to them.

The term apraxia introduced by Kussmaul, is not exactly synonymous with mind-blindness or soul-blindness, but includes this affection and more. As defined by Kussmaul, apraxia is the loss of the memory of the uses of things and the understanding of the signs by which things are expressed. Usually these are matters of visual memory and the signs usually become known to us through sight, hence mind-blindness and apraxia in such cases signify the same thing; but the patient may be apraxic through other senses than sight; if, for instance, he is unable to recall through hearing the nature of the object or objects which emit the sound, he is mind- or soul-deaf,—that is, he is apraxic through the sense of hearing. The rustling of leaves or the sound of a waterfall, or the notes of a bird may fail to recall the objects producing the sound, just as in the word-deaf, the sounds of words convey no meaning and in the music-deaf, musical tones are unrecognized as such. Tactile apraxia is exhibited when the patient is unable to recognize the nature and use of an object through touch. In this section we are concerned with apraxia only so far as it relates to sight.

Burr[49] reports the case of a woman sixty years old who, while sitting at the table, suddenly lost vision. This patient, when examined by Burr, was mind-blind, and in addition she failed to recognize objects with the hands. She could not, for instance, tell what a watch was when placed in her hands, and she could not feed herself because of her inability to recognize a spoon, knife and fork. She seemed to recognize the hand of another person when it was grasped and also a few other objects. Here the apraxia was through touch and perhaps stereognostic perception as well as sight. Weir Mitchell[50] had recorded a similar case in 1892.

What has just been said regarding apraxia refers to sensory apraxia, —that is, to the disorder due to disease on the sensory or receptive side of the brain. Cases are on record, however, which seem to show that a motor as well as a visual or other form of sensory apraxia may be exhibited, and it is worth while to briefly refer to these cases as they belong to the matter in hand in a diagnostic way because it may sometimes become necessary to differentiate motor apraxia from visual or other type of the sensory disorder. Pick,[51] for example, has recorded a remarkable case which he believes to be one of motor apraxia. A man sixty-two years old had for three years before coming under observation, peculiar attacks. He would suddenly find himself unable to speak the words which he wished to utter or to do anything desired. He could not read or write, and everything seemed to swim before his eyes. He had neither paralysis nor loss of hearing, and in three days he was again normal. Similar attacks occurred about once in two weeks, and were associated with slight tonic masseter spasm. On one occasion he saw a mist and fiery rings.

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[49] Burr, Jour. of Nerv. and Ment. Disease, May, 1897.

[50] Mitchell, Medical News, vol. lxii. Oct. 1892.

[51] Pick, Neurol. Centralbl., vol. xxi. Nov. 1, 1902.

One of the most remarkable cases of apraxia ever recorded has been reported by Liepmann.[52] In this case the disorder was unilateral; the patient was neither hemianopsic nor mind-blind. The tactile and pain senses were much diminished in the right half of the body, especially in the right limbs. The power of localization and the sense of position and of movement of the limbs seemed to be absent. The only motor paralysis was in the lower branch of the left facial nerve. This patient had motor aphasia and could only say a few words, these being always the same; in other words, they were the recurring utterances of Hughlings Jackson. He understood what was said to him, could read, and obeyed simple commands when they were written, provided his movements were not performed with the right limbs alone. The movements of the head, including those of the face and tongue, were apraxic on both sides. When he was asked to make any movement with his right hand, such as touching the nose with the finger, his attempts were unsuccessful; but with the left hand the command was promptly obeyed. When he was tickled in the right ear he made no movement of defense; but with the left hand he tried to remove any object that was placed in his left ear. He was unable to remove with his right hand a needle stuck in his right thigh. When asked to pick up with his right hand one of several objects placed before him, he was unable to comply with the request. It is probable that he understood what was asked of him, because after he had failed to pick up with the right hand the desired article, he was able to pick it up with the left hand when simply told to use this hand, without the name of the article being mentioned again. With the right hand he was completely agraphic; but with the left he was able to do mirror writing, although the letters were very irregular. His gait was normal. Occasionally he performed an intentional movement correctly.

In this case the patient was supposed to be suffering from aphasia with dementia, a fact of medicolegal as well as of neurological interest. Liepmann believed the lesion existed in the third frontal gyrus and insula, without implicating to any extent the motor area, or the inferior or superior parietal convolutions. It is difficult, however, to believe that the lesion was so placed, or at least so limited, as I have seen and reported a case of destructive lesion of the left third frontal and insula, in which no such unilateral apraxia was present although the case presented typical aphasia.

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[52] Liepmann, Monatschrift für Psychiatrie und Neurologie, July, August, and September, 1900, vol. viii. This case is cited by Spiller, with comments, in Progressive Medicine, 1900–1901.

The discovery of the symptom known as mind-blindness was due to the physiologist Munk, who had observed it as the result of several extirpation experiments on dogs. He found in some cases, when he removed a portion of the occipital lobes, the dog, while still retaining the power to see objects, and to avoid them through the medium of sight, was evidently unable to recognize the meaning and use of such objects. The dog from which a portion of the occipital lobe had been removed, for example, no longer recognized his master, other dogs with which he had been long familiar, or the food which was placed before him,—except, in the last case, through the sense of smell. He gave to this symptom the name of *seelenblindheit* (soul-blindness or mind-blindness). He believed that this symptom was due to lesion of the region for visual memories, the visual memory field, the visual portion of the concrete memory field or concrete concept area of the writer. Flechsig holds practically to the same view, believing that not only mind-blindness but mind-deafness are due to lesions of portions of this great association area.

The truth is that all symptoms which result from impairment or disturbance of concrete concepts or concrete memories are to be sought for in lesions of centres located in the great concrete concept area, or in the tracts going to or coming from the centres of this area, or associating them together. Mind-blindness results, or may result, from lesion of the visual cortex or subcortex of this area; mind-deafness, from the auditory portion of this area; astereognosis, from lesions of that portion of this area devoted to the muscular sense and stereognostic perception; tactile amnesia, from lesions of the centres and tracts concerned with tactile recognition; music-deafness, from lesions of the centres and tracts related to the function of intonation; and so on throughout the entire list of concrete memories and their centres and tracts.

Munk's views regarding the extent of cortex sufficient to cause mind-blindness do not appeal to the writer, as they have not to others, as correct,—this for theoretical reasons and because of clinicopathological observation. Munk expressed the opinion that extirpation of the circular area of the cortex so small as fifteen millimetres in diameter and two millimetres in thickness would cause in the animal the phenomena which he designated as *seelenblindheit*. The term *seelen-*

blindheit was used by Munk in contradistinction to *rindenblindheit* or cortical blindness, in which the animal becomes totally blind to all impressions; it is unable, in other words, to see any objects presented to it as well as to recall the image of one formerly seen. Observations on man show that, whatever may be the case with dogs, it is necessary that a somewhat larger area of the cortex on both sides shall be destroyed than that indicated by Munk; and yet he was not as far wrong as Goltz and some of his most caustic critics would have us believe. Cases are now on record, some of which will be presently given, in which comparatively limited lesions of the visual cortex of both hemispheres have given rise to mind-blindness. It is not, however, correct to regard the regions, destruction of which causes mind-blindness, as those for clear or central vision; while, as has been stated, the centre for the macula is the centre for clear vision and for the recognition of minute objects and objects with special forms, such as those for words, letters, numbers and geometrical figures, the cortical areas of peripheral representation, like the peripheral retina itself, are capable of recognizing many objects. The truth is, as I have already stated, that mind-blindness itself needs division and has a considerable sub-divisional cortical representation. The individual may be mind-blind for persons, or places, or figures, words or letters, or any of these or any combinations of these, the powers of recognizing the others remaining.

One of the most important of the early cases of mind-blindness is that recorded by Lissauer [53] in great detail and with a discussion by him of the pathology and symptomatology of the affection and the methods of studying it. The patient was a man eighty years old, who for three years before coming under observation had had attacks of dizziness which were often so violent that he fell. For about a year he had been forgetful; he often could not recall, for instance, the ages, names and number of his children. After a special attack he complained that he could not see as well as before. He bumped against doors and other objects and could not go around alone. He mistook parts of his dress and all sorts of objects; he did not recognize his own daughter. His mind was clear for familiar business matters. The attacks of mind-blindness were transient.

On examination, while he showed signs of old age, he was not in any way demented. While he could speak intelligently and easily he was unable to recognize everything. By touch and hearing he at once recognized everything. His sight, except for his mind-blindness, was good. He drew objects or designs but afterwards he could not recognize them. He perceived but could not apperceive. With the exception of the mind-blindness, nothing else of importance could be made out. His hearing was dull, but he showed no cerebral affection of hearing or of speech. He was not paralyzed and had no impairment or disturbance of sensation. His ocular movements and reflexes were good.

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[53] Lissauer, Archiv für Psychiatrie und Nervenheilkunde, vol. xxi. 1889–90.

The patient during six months after he first came under observation made some improvement, and then failed to make any further progress. At times during this period considerable vacillations were observed in the amount and degree of his mind-blindness. He would recognize objects at one time while he would fail to do so at another. Even after he had made considerable improvement he would occasionally show a degree of inability to recognize objects as bad as he had at any time. The case was not followed until death but is nevertheless one of much value because of the careful manner in which it was investigated and described.

In a case reported by F. Müller,[54] the left occipital lobe was almost destroyed by a tumor and softening, and the posterior part of the callosum was implicated in the softening, and the associating tracts connecting the right occipital lobe with the left hemisphere and its speech centres were interrupted. In this way Müller explains the optic aphasia and alexia which were present. Mind-blindness was also present in this case.

Müller mentions that mind-blindness is often associated with optic aphasia and asymbolia, and he gives the reference to a number of cases. Even more commonly it is associated with alexia, and he refers to several examples. In a number of cases right hemianopsia existed. Müller says that in all cases of pronounced mind-blindness with satisfactory necropsy (1892) disease of the occipital lobe was found, or of the parts adjoining, as in two cases in which the parietal lobe was affected. Of twenty cases, both hemispheres were diseased in fourteen; and of these both occipital lobes were affected in twelve; in one, the right occipital lobe and the left frontal lobe were affected; and in one the left occipital and the right parietal lobes were affected. In six cases the lesion was unilateral, and of these the left hemisphere was diseased in four cases (in two the left occipital and in two the left parietal). In two cases the right occipital lobe alone was diseased.

Wernicke, Wilbrand, Bruns, Stolting and others believe that the occipital lobes are of equal value in function, especially in the recognition of objects, and that mind-blindness can exist only when both occipital lobes are diseased. Müller thinks it has been shown satisfactorily that mind-blindness may be caused by unilateral lesion, as the examination of the brain must be microscopic and not merely macroscopic. It seems to him more probable that lesions of the outer portion of the occipital lobes, especially on the left side, cause mind-blindness. The destruction of the tracts in the callosum connecting the occipital lobes or one occipital lobe with other parts of the opposite hemisphere is of much importance in the causation of mind-blindness.

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[54] Müller, Arch. für Psych., 1892, vol. xxiv. p. 856.

Liepmann[55] has reported a case of practically uncomplicated mind-blindness in a music teacher, aged fifty years, who had an apoplectic attack, September, 1901. After this he did everything in a reverse or foolish manner as, for instance, washing in his soup. He did not recognize large objects, nor his own reflection in the mirror, but recognized persons by the voice. He did not know his own wife by sight; color perception was much disturbed. A small defect was present in the upper right quadrant of the visual field; vision was two-thirds. He could imitate large movements fairly well. Well-known objects were often wrongly named. Recognition through touch was slightly disturbed, but through hearing was normal. Motor aphasia was absent, as was also sensory aphasia, unless his wrongly naming objects might be so considered.

The patient died in 1904, two years after the case was first recorded by Liepmann. A necropsy showed most interesting results. Lesions were found on both sides of the brain, on the left in the lower occipital convolutions, and on the right in the angular and second occipital convolutions. In the right hemicerebrum the area of softening was on the convexity, and extended from the angular gyre through the gray and white matter of the second occipital gyre.

A case reported by Sérieux[56] is of value notwithstanding the complication of object-blindness with other symptoms like word-blindness. This case, which was published in 1891, is cited by Wyllie. The patient was a woman sixty-two years old, who in 1888 had a temporary paralytic attack with lasting visual disturbances, and in 1890 had a second seizure with epileptiform spasms, hallucinations of hearing, and delirium. When examined six months later, she was word-blind, agraphic, word-deaf, and paraphasic. She also showed marked mind-blindness with intact general intelligence. She was unable to recognize her nearest relations, said that people around her seemed to be wearing masks, that her daughter's face appeared " flat as a skate." The object-blindness was most marked with regard to faces, although exhibited in less degree with reference to other things. She could recognize some objects, but not others; a comb, for instance, was mistaken for a pen holder. She died of pneumonia a few months after the observations of Sérieux were made. At the autopsy, lesions were found in both hemispheres. In the left hemisphere were present patches of softening in the supramarginal (subparietal) gyre, and in the caudal extremity of the first temporal gyre; in the right were large areas of softening involving the angular and supramarginal gyres, and also the caudal extremities of the first and second temporal.

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[55] Liepmann, Allgemeine Zeitschrift für Psychiatrie, Berlin, vol. lix, 1902.

[56] Sérieux, Compt. Rend. Soc. de Biol., December, 1891.

So far as is known to the writer, the few other cases of mind-blindness which are recorded are not of equal value with those of Liepmann and of Sérieux, but all point to the existence of centres for object recognition by sight in the temporo-occipital convolution from the second to the fourth or fifth.

Two cases have fallen under my observation, of transient or recurring mind-blindness. In both of these cases operation was performed for the removal of a brain lesion, presumed to be a tumor, although in one case at least the result did not confirm this opinion. In this case the patient presented in the right half of the body disorders of sensation, stereognosis and co-ordination. He was partially word-deaf, word-blind, and letter-blind, and late in the case showed marked hemiparesis. The operation was performed with the view of exposing the brain at the posterior extremity of the first temporal convolution. A large patch of necrotic tissue, having somewhat the shape of a pyramid, with its base towards the occipital pole of the brain, was uncovered. The lesion may have extended further than was revealed by the operation. It not improbably included the caudal extremities of the first and second temporal convolutions, and possibly more.

The operation was performed four years prior to the writing of this article, and the patient is still living and in fair health, but is hemiplegic, partially hemianæsthetic and aphasic. He suffered at times from curious attacks which can properly be described as periods of mind-blindness or object-blindness. Recently, I re-examined this patient. I found that his hemiplegic condition had gradually become worse, the paralysis having visibly increased, especially during the last year. He showed some slight improvement in the ability to name objects looked at, also in being able to name many words. He could not, however, form phrases or sentences. He seems still to be partially word-deaf, being thrown into confusion in trying to understand much that was said to him. He continued largely word-blind, but recognized an occasional word, writing being understood rather better than printed matter.

His wife, who accompanied him, stated that, beginning about two months after the operation, the patient had at irregular intervals spells of temporary mind-blindness or object-blindness. In these, which would last from a few minutes to one or two hours, while he would seem to know his wife, he was apparently unable to recognize others. In these attacks also he appeared unable to recognize objects or his surroundings; he seemed dazed, would look around as if wondering who the persons and what the objects around him were, repeating almost constantly one word, " What, what." He could not understand anything that was said to him at the time of these spells, and became greatly confused and irritated. His wife said that he remembered these spells after they passed off, and would say " Awful, awful," as if in great dread of them. Examination showed that he understood the use of most objects, such as the telephone, a pen, ink, shears, and paper. An effort was made to examine him for color-blindness; he could not recognize red and blue, but seemed to know green and yellow; but when the examination was continued, he became confused, and was unable to recognize any colors.

In the second case referred to, in which operation exposing the occipital lobe was performed, and portions of the cortex removed or contused, corresponding to the parts of the first and second occipital convolutions, the man suffered not only from optic aphasia, but temporarily from object-blindness. At first, he did not at times recognize persons and things. The impairment was only partial.

Briefly stated, the pathological cause of mind-blindness is, in the first place, a lesion or lesions of the visual portion of the concrete memory field. Usually the disease is on both sides of the brain, but transient forms of the affection, and perhaps, in exceptional instances, the persisting disorder may be caused by a destructive lesion of the left optic memory field. In the second place, lesions of the tracts which connect the higher visual field with the lower or primary visual centres may cause this symptom. When the affection is due to a lesion of the association fibres it is, if unilateral, so situated as to destroy the tracts which connect the primary or lower visual centres on both sides with the higher visual area on one side.

WORD-BLINDNESS AND ALLIED DISORDERS.

In several places in the present chapter reference has been made to the fact that in the angular convolution of the left cerebral hemisphere and its immediate neighborhood is situated a centre for the storage of the visual images of words. It is not infrequently spoken of as the centre for word-seeing. It is the last and most important development of the higher visual area, the visual portion of the concrete memory field. A destructive lesion of the cortex of this region or of the tracts entering it from the primary or lower visual centre will cause the symptom to which Kussmaul gave the name, word-blindness. One of the best definitions of word-blindness is that of Bateman, who describes it as a form of verbal amnesia in which the patient has lost the memory of the conventional meaning of graphic symbols. He sees these symbols, he knows that they differ from their environments, but he cannot translate them mentally into words. They are to him as if they had never had any significance.

The two chief varieties of word-blindness now usually described are known respectively as cortical word-blindness and pure word-blindness. By cortical word-blindness is meant that variety of this form of aphasia which is due to lesion limited to the cortex; while the term pure word-blindness was applied by Dejerine to the form which is dependent upon a lesion of the tracts which enter the cortical centre for word-seeing from the primary visual centres. The term pure word-blindness like pure word-deafness does not seem to be a happy one, but is perhaps better retained, as it has become fixed in the literature of the subject. It is objectionable in the sense that a number of similar designations are of doubtful clearness,—as, for instance, Munk's soul-blindness (*seelenblindheit*), which is used as a contrasting term with his cortical blindness (*rindenblindheit*): one designation is founded upon the psychological idea, that of psychic blindness, and the other is anatomico-pathological in its origin, referring to the blindness caused by the destruction of cortical brain substance.

In cortical word-blindness the patient is unable to comprehend words by sight and is also agraphic. Owing to the loss of the memory of visual images, he cannot recall these for the purposes of writing. In the pure word-blind patient, while he cannot comprehend words, either printed or written, by looking at them he may be able to write spontaneously or from dictation, as his cortical centre for the visual images of words and letters is not destroyed, but if his eyes be removed even for a moment from the written page he cannot recognize the word which he has just written.

While we owe to Dejerine a clearer conception of the manner in which word-blindness usually originates, the observation of two forms of word-blindness, one in which the patient is agraphic, and the other in which he retains the power of writing from dictation or spontaneously, is not new. Westphal, who is cited by Kussmaul, many years ago recorded the case of an actor who was hemiplegic and incompletely aphasic. Although he could speak with fluency, he could not recognize or understand some words. He was completely alexic, and while he could write from dictation, he immediately afterwards could not comprehend the words he had written.

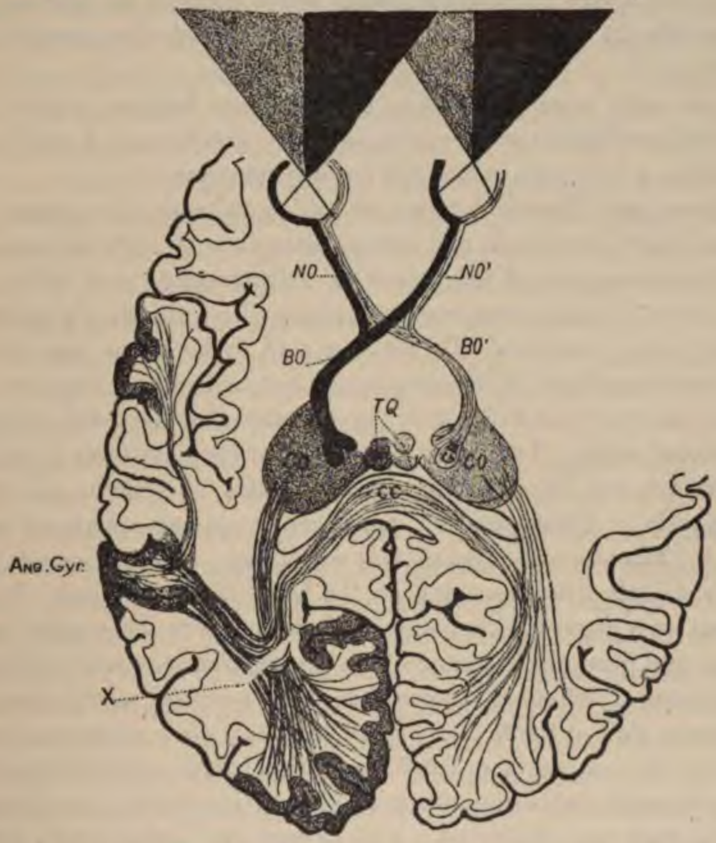

Fig. 18.—Dejerine's diagram showing the position of the lesion causing pure word-blindness.

The exact view of Dejerine with regard to the manner in which pure word-blindness is caused can be best shown by a reference to the diagrammatic illustration, Fig. 18, which is reproduced by permission from Wyllie's work on the Disorders of Speech.

"An examination of this diagram (Fig. 18) will enable the reader to understand quite easily the relation of the eyes to the primary visual centre in the occipital lobe. In the diagram, the left optic tract and its connections are, for the sake of distinctness, given in deep black. The reader will, on looking at the diagram, at once understand how section of the left optic tract, BO, would produce homonymous hemianopsia in the opposite half of the field of vision. He will also see that the same effect would be produced by a lesion further back, either in the optic thalamus, in the radiations of Gratiolet, or in the primary cortical centre itself. But he will note that word-blindness will not, in any of these cases, be added to the hemianopsia, unless the lesion be situated at about x, and so cut across not only the fibres which connect the angular convolution with the left occipital lobe, but also those which connect it with the right one."

As the early cases illustrating the difference between cortical and precortical word-blindness are comparatively well known, I shall refer here only to a few cases somewhat recently reported.

Dejerine and Thomas[57] report an additional case of a woman who developed right hemiplegia and hemianæsthesia with right hemianopsia and simultaneous loss of the power of writing eight years before her death. After a slight temporary paraphasia she regained the power of speaking; she understood all that was said to her; she was able to write, but immediately afterwards could not understand what she had written; she was word-blind, with the exception that she could recognize an occasional letter. Two years after her first apoplectiform attack she had a second, and after this she became unable to write at all, either spontaneously or from dictation, her other symptoms remaining much the same. The lesion in this case was very large; so large, indeed, as to interfere a little with reasoning on the basis of the findings. It was important with regard to the views of Dejerine as to the cortical word-blindness and pure word-blindness that the angular convolution escaped. A large focus of softening involved the third temporal convolution and the posterior ends of the first and third occipital, while ventromesially it destroyed the cuneus, the fusiform, lingual and subcallosal gyres, the hippocampus and cornu ammonis; the pulvinar and internal geniculate body were also destroyed. The patient died from recent hemorrhage.

One of the most recent cases bearing upon the subject of pure word-blindness is recorded by Hinshelwood, Macphail and Ferguson,[58] this report also containing several other facts of great interest to the student of aphasia. A man, fifty-three years of age, who was a teacher of French and German, suddenly found that he had lost entirely the ability to read words or letters. He could write correctly to dictation but could not, a short time afterwards, read what he had written. He had a right lateral homonymous hemianopsia. He was under observation for nine years until his death, no other symptoms developing. He was able, only to a very limited degree, to reacquire the visual memories of words. His death was due to a hemorrhage into the right half of the cerebellum as was shown by necropsy. The lesion causing the word-blindness, letter-blindness and hemianopsia was a necrotic cyst in the occipital lobe. The cortex destroyed was summarized by the recorder as a large, triangular area limited by the calcarine fissure internally and by the third temporal gyre externally, with the apex at the occipital lobe. The cortex of the cuneus with the exception of the posterior portion of the calcarine fissure was not destroyed; the fibres passing to and from the cuneus were, however, involved as were also the fibres going from the cuneus to other portions of the occipital cortex, and also the fibres passing to the left angular gyre from both the left and right primary cortical visual centres. As the left angular gyre was intact, it will be seen that the case, as the authors assert, supports the views of Dejerine and of Sérieux as to the distinction between precortical or pure word-blindness and cortical word-blindness.

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[57] Dejerine and Thomas, Rev. Neurol., July 15, 1904, p. 645.

[58] Hinshelwood, Macphail, and Ferguson, Brit. Med. Journ., Nov. 12, 1904.

I have emphasized the views of Dejerine with regard to the distinction between his pure word-blindness and cortical word-blindness, or what I would prefer to call precortical or qualified word-blindness, and cortical or absolute word-blindness, and also have given his hypothesis as to the position of the lesions in these two forms of visual aphasia illustrating these facts by references to a few recorded cases. It must not be understood, however, that the views of Dejerine necessarily explain all cases of his so-called pure word-blindness. Cases are on record which do not bear out the supposition that this form of visual aphasia is always dependent upon a precortical lesion. Several cases are now on record in which the so-called pure word-blindness or pure word-deafness or both, in the same case, were present, yet the lesion in whole or in part affected the cortical centres for word-seeing and word-hearing. Strohmayer[59] has examined the cases of pure sensory aphasia accessible to him, six in all. He points out the important fact that in five of these cases the lesions were bilateral, implicating both the cortex and the subcortex of the temporal lobe. In only one case was the lesion unilateral, and here it was confined to the left temporal cortex. From a study of these cases he argues that the lesions causing pure sensory aphasia are bilateral, although, as has been indicated, a few cases like those of Dejerine and Sérieux, for instance, seem to make this doubtful.

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[59] Strohmayer, Deutsche Zeitschrift f. Nervenheilkunde, vol. xxi. Nos. 5 and 6, p. 371.

As has been shown in a preceding section of this chapter, according to the views of Lannegrace, Ferrier, Gowers and the writer, each angular gyre is to a certain extent a centre of representation for both maculæ. If this be the case, it might be possible for the patient to be word-blind from lesion of the higher visual area of one hemisphere with or without a precortical lesion or a lesion in the opposite hemisphere and yet retain some power of writing spontaneously and from dictation with the help of the undiseased angular convolution.

Hinshelwood [60] recently reported a case which seems to show that when an individual knows more than one language he may become word-blind to one language alone, or the word-blindness may vary in degree in the case of the different languages. He had a patient who became unable to read English, but could read Greek, and had partial word-blindness for Latin. On testing this man for French, a greater degree of word-blindness was manifested than in the reading of Latin. Even with English the word-blindness was not absolutely complete, and there was no letter-blindness. Hinshelwood believes that in the case of a person who is able to read several languages, the letter and word visual images of each language will be grouped together, forming thus a series of separate groups within the centre for word-seeing. If the whole centre is destroyed or its functional activity abolished, the individual will be word-blind to all languages; if there is only a partial interference with the centre, then the word-blindness may be only partial and may be limited to a single group of these visual changes,—*i.e.*, to a single language. The same writer refers to another patient who was word-deaf to French, his native tongue, but could understand what was said to him in English. The explanation offered is similar to that given for his case of word-blindness to languages; in other words, if in the word-hearing centre, the auditory images of different languages are arranged in separate groups, in a partial lesion one group may be interfered with or destroyed, while the others are preserved.

Usually in word-blindness as in any other form of sensory aphasia or of motor aphasia, the lesions determining the symptomatology are located in the left hemicerebrum; the only exceptions to this presumably being those cases in which the cerebral affection occurs in left-handed persons. Of the few cases which have been seen to confirm this left-sided localization the right-handedness of at least some of the patients affected with the aphasia has been in doubt.

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[60] Hinshelwood, Lancet, Feb. 8, 1902, p. 358.

Recently in conjunction with Dr. T. H. Weisenburg, I have published the account of a case of marked sensorimotor aphasia associated with left hemiplegia and some other phenomena.[61]

The patient at the age of thirty-five had a severe apoplectic stroke. He was seen by me a day or two after the seizure and then presented the usual phenomena of a severe cerebral hemorrhage. When he recovered from the immediate effects of the attack, he was found to be completely hemiplegic on the left side and almost absolutely aphasic. He was seen by me only once or twice at times closely related to his apoplexy. I saw him, however, a year later when he regained the use of his left extremities so that he was able to walk about with the usual hemiplegic gait, and his aphasia had improved to a certain extent.

About a year after his first seizure he had a convulsion, three or four the following year, and the third year still more. Dr. Harvey Cushing, of Johns Hopkins University, performed an osteoplastic operation designed to completely cover the motor region of the right side, and to a less extent, the area posterior to it. After the operation, up to the time of his last coming under observation, which was about a year later, he had one or two spasmodic attacks. These attacks were mostly, if not altogether, left sided.

Four years after he was first seen by me in his acute apoplectic condition, this patient returned and remained under my care for about two months, during which time systemic efforts were made to improve his aphasia by training, Dr. Weisenburg being associated with me in this work. At this time a careful study was made of his cerebral symptoms. In the first place, he still had marked hemiplegia, the leg, arm and face being affected, although the last to only a slight extent; the deep reflexes on the paralyzed side were exaggerated and a typical Babinski was present; on the entire left half of the body he was hypæsthetic to touch, pain, temperature and to vibration sensation (the sensation which results from the placing of a tuning fork on bony or other points); sensation of position was partially abolished in the left hand, in which he was also largely stereognostic; the ophthalmoscope showed a normal fundus and partial left lateral homonymous hemianopsia. He was not word-deaf, or, if so, only to a very limited degree; he understood what was said to him with perhaps rare exceptions. He could repeat anything that was said to him, his articulation and enunciation being excellent.

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[61] Mills and Weisenburg, Medicine, 1905.

His most interesting aphasic phenomena were in the visual sphere. Immediately after his attack he had been undoubtedly word-blind and letter-blind. At the time when the careful examinations were made, his word-blindness and letter-blindness still remained in very large degree. He was not, however, object-blind, distinctly recognizing persons, and also the use and nature of objects around him. He knew a few letters as, a, b, d, o, s, which he had learned as the result of many months of teaching. Even the letters which he knew he could not pick out from the midst of the word in which the letters were present. As the result of prolonged teaching, he could only recognize a few words, and these were chiefly those with which he had been extremely familiar in his past life. He could, for instance, pick out the words *apple* and *seed*, he having been in a business in which such words were frequently used, both by mouth and by written signs. He could sign his own name or he could write his own name with the appended words "and wife;" if what he had written was presented to him later, however, he seemed to be utterly oblivious of its meaning. He had learned to write his own name with the added words "and wife" in order to register at hotels. He could do this as well with his eyes closed as open. He was an alexic and visual agraphic.

Close investigation of this man's history seemed to show that he was born right-handed. He ate, wrote, and did by preference almost everything with his right hand. When he was four years old he broke his right arm, and during the time that he was unable to use it, he learned to do many things with his left hand. He would, for instance, play ball with his left hand by preference. This tendency had apparently arisen out of the conditions of the fractured right arm.

He did not write with his left hand, and was not, in the ordinary sense of the term, ambidextrous. We can assume, therefore, that the patient was congenitally and by acquired habit a right-handed individual. The lesion in this case was undoubtedly on the right side, and was either a subcortical hemorrhage involving the tracts going to the precentral, postcentral, superior parietal and angular convolutions, and possibly the upper part of the first temporal convolution, or an embolus of the right middle cerebral artery. The optic radiations may, to some extent, also have been involved.

The important features of this case were (1) that in a right-handed man with left hemiplegia aphasia largely of a sensory type appeared and this aphasia was persistent; (2) the aphasia consisted principally in word- and letter-blindness, and this was due to a lesion in the right side of the brain.

Instances of crossed aphasia, the term first applied by Byrom Bramwell, are very rare. By crossed aphasia is meant aphasia which is, so to speak, against the rule. As is well known, aphasia usually occurs from a lesion of the left hemisphere and in association with right-sided paralysis, but in crossed aphasia the symptom is due to a right-sided lesion in a patient who is right-handed, or a left-sided lesion in a person who is left-handed.

Senator,[62] who recorded one of the most recent cases in 1904, was able to collect only eleven cases from literature, one or two of which are somewhat doubtful; and in all, with the exception of Senator's case and the one we now report, the hemiplegia was right-sided and occurred in left-handed individuals.

The causes of crossed aphasia may be explained in several ways: (1), the lesion may involve the cortex of both sides; this, however, is exceptional and was not present in any of the recorded cases; (2), there may be no decussation of pyramidal fibres, the lesion being on the same side as the hemiplegia. This is exceedingly uncommon, having only been recorded in a single case; (3), the lesion causing aphasia in a right-handed individual with left hemiplegia may be on the right side, and in a left-handed person with right hemiplegia on the left side.

It is not necessary to consider the first two hypotheses in the case just given, for a lesion of the right side undoubtedly caused the left hemiplegia and aphasia. It is difficult, however, to explain why the centre for word- and letter-seeing should be situated on the right side in a right-handed individual.

The question, of course, arises as to what factors determine on which side of the brain the motor speech centres and the more specialized centres, as of word- and letter-seeing, will be localized. All things considered, in a child which has not as yet learned to write or read, heredity, congenital tendency and acquired habit determine whether the child shall be right- or left-handed. We can assume, therefore, that a child whose parents are right-handed and in whom there is no physical deformity to prevent the use of the right hand, will be right-handed, and that its leading speech centre (presuming that both sides of the cortex have to do with motor speech) will be located on the left side in Broca's convolution. The converse, of course, is true in left-handed persons.

This, however, is by no means always the case. Weber[63] records instances of children who had not as yet acquired the ability to write, in which a lesion on the right side would cause aphasia in right-handed persons, and vice versa. In other words, in children, or for that matter, in adults, who had not as yet learned to write, Weber assumes that the motor speech centres are potential on both sides of the cortex. This is probably so, for it is improbable that the speech centres are equally divided between the two sides of the cortex, even in childhood. Here heredity, congenital tendency and acquired habit will probably make the child either right- or left-handed, and therefore right- or left-brained.

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[62] Senator, Charite annalen, vol. xxviii. 1904.

[63] Weber, Centralbl. f. Physiol., xxiii. Nr. 12.

The factor which determines on which side such highly specialized centres as those for word- and letter-seeing would be localized is probably the art of writing. This, no doubt, also determines on which side the leading motor speech centres will be. This was well shown in a patient of Bramwell.[64] A man of thirty-six, who was left-handed for everything except for writing, had right hemiplegia with persistent motor and sensory aphasia, evidently from a lesion in the left hemisphere.

A case of Oppenheim[65] also illustrated this point. A woman of fifty-nine, who, since she was seventeen, had been left-handed because of an injury to the right hand, developed complete motor and sensory aphasia, due to a tumor in the right cerebral hemisphere.

In the patient whose case is just recorded, however, the man always had been right-handed and had always written with his right hand. At the age of four, as already stated, he had broken his right arm and had learned to do many things with his left hand. Even for those purposes for which he commonly used his left hand, he could and sometimes did employ the right. We must assume, therefore, that although he wrote with his right hand and was born and continued right-handed, except as stated, his motor and sensory speech centres were situated in the right hemicerebrum. On inquiry it was learned that the patient's parents, grand-parents and collateral relatives were all right-handed. It must be assumed, therefore, that in exceptional cases, in spite of the usual laws of heredity and of acquired tendency, there is, for some unknown reason, an inclination toward the localization of the motor and sensory speech centres in that side of the brain on which they would not ordinarily be expected.

It is also highly interesting to note that in the case discussed the aphasia [illegible] confined to word- and letter-blindness. Köster[66] records a case with necropsy in which, with left hemiplegia in a left-handed person, there was word-blindness, and in addition, left hemianopsia. At the necropsy extensive destruction of the right cerebral hemisphere was found. Köster believes that this was the first case reported with necropsy, of word-blindness due to lesion of the right hemicerebrum. This case resembles the case under consideration to a large extent, except that the patient was right-handed.

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[64] [illegible] The Lancet, June 3, 1899.

[65] [illegible] f. Psych., vol. xxi. 1890, p. 139.

[66] [illegible] Centralbl. f. Nervenheilkunde and Psychiatrie, February,

The case here recorded is also instructive from another standpoint, for it teaches that not only in exceptional cases may the centre for letter- and word-seeing be situated on the right side, but also that a unilateral lesion may cause permanent loss of word- and letter-seeing, opposing the views held by Bastian and others that these functions have a bilateral representation.

Joffroy [67] records the case of a right-handed man, aged 77 years, who was suddenly seized with mental agitation, after which he presented almost complete word-deafness and word-blindness. He could recognize some letters and could appreciate the meaning of certain words. Paraphasia, paragraphia, and sometimes jargon utterances were present. He could write his name with difficulty, but could not copy nor write from dictation. Death occurred from cerebral hemorrhage three months later. Examination of the brain showed that the left hemisphere was normal. The right hemisphere presented a patch of softening involving part of the posterior third of the first and of the second temporal gyres. Around this patch shrinking of the convolutions and adherence to the membranes were present, extending over the posterior two-thirds of the first and second temporal gyres and involving the whole angular convolution.

Joffroy believed that this man was congenitally left-handed, but that he had become trained to be right-handed, and he offers this explanation for the situation of the speech centres upon the right side of the brain. No ancestral left-handedness is mentioned in the history.

Letter-blindness (literal blindness, amnesia literarum) is a form of blindness due to cerebral cortical or subcortical disease in which the patient is unable to recognize letters, although the visual apparatus, from the periphery to the primary cortical visual centre, or cortical centre for optical perception, and this centre itself are intact. Letter-blindness may exist without word-blindness, and word-blindness without letter-blindness, although both are usually associated. Curious facts regarding blindness to letters from lesions of the cortical centre for higher visualization, or the entering tracts to this centre, are often observed. The patient may retain the ability to recognize one or two or several letters of the alphabet, but fail to comprehend all the rest. We believe that letters and words are represented in separate cell groups of the cortex, although these are adjacent.

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[67] Joffroy, Rev. Neurol., Jan. 31, 1903, p. 112.

Alexia, the inability to read because of cerebral disease or disturbance, may be due to lesions variously situated. In its most complete and typical form it is dependent, as has already been shown, upon a lesion of the cortical centres for word-seeing and letter-seeing of the tracts which pass to these from the optic perception centres or primary cortical centres for sight in the calcarine cortex. Some degrees of alexia may, however, be present from a lesion of the centres for word-hearing, naming, or of Broca's centre, as well as of the tracts which connect these various centres. All of the centres in the zone of language take some part in the verification of the correctness of the varieties of speech spoken and written.

It is held by some writers on aphasia that if the word-hearing centre is destroyed the patient will be unable to read. My own experience, however, shows that this is not always the case, and Wyllie and others give cases in which word-deafness was present without alexia. In these cases reading is probably made possible by direct connections which exist between the visual centres for words and letters and Broca's centre.

In so-called motor aphasia from lesion of the left third frontal and insula, alexia in some degree may be present, but it is certainly not absolute. In one of my cases which was under observation during a period of many years, I was able to demonstrate in various ways that the patient was able to read silently, although his reading may have been imperfect. When, however, he attempted to read aloud, he always repeated the same words. The visual images in this patient had the power to revive the auditory images, although perhaps not perfectly. Necropsy in this case revealed a destructive lesion confined to the left third frontal and insula.

Paralexia, in which the patient transposes or substitutes words and syllables in reading, is caused by lesions variously situated in the zone of speech, as by partial lesions of the visual centres, by lesions of the auditory and motor speech centre and of the tracts connecting these centres with the visual centres for language. Doubtless there is a silent paralexia, the patient in some cases transposing silently.

Dyslexia, or fatigue in reading, the result of lesion or disturbance of the cerebrum, is sometimes observed. The patient may be able to read correctly either script or text, to read aloud or to repeat what is read by others, but the effort soon becomes fatiguing, the reader tires and may be unable in a short time to go on with the reading. It is a condition comparable to that which is observed in some cases of word-deafness which have partially recovered, and is sometimes associated with partial word-blindness. In one such case of word-deafness recently observed by me, the patient could listen attentively and understandingly to one person quietly talking to him for a short time, but if the conversation was continued, and especially if it became more varied, his attention soon tired and before long he would entirely fail to appreciate what was said to him. If several persons were talking to him at the same time, he also became confused and unable to comprehend what was being said. Reading and writing of an imperfect sort could be continued for a short time, but even these became impossible after a few minutes.

Dyslexia may be due to a partial lesion or a functional disturbance of almost any portion of the zone of speech. In its most typical forms the lesion or disturbance is usually in, or in close connection with, either the visual, the auditory, or the motor speech centre. It is sometimes a premonitory symptom in apoplexy, doubtless indicating some ischemia in a region which subsequently becomes the seat of hemorrhage or occlusion.

Agraphia and allied conditions, like dysgraphia and paragraphia, may be due, like alexia, dyslexia and paralexia, to lesions variously situated in the zone of speech, as in the centre for the visual images, that for auditory images, Broca's centre, and the so-called motor-graphic centre in the posterior part of the second frontal convolution. More or less complete agraphic disturbance may also be due to lesions of the tracts connecting these various centres. We are here, of course, chiefly concerned with visual agraphia, that form of impairment of reading which is dependent upon lesion of the visual centre for language or the tracts which lead to or from it.

Stanley Barnes[68] has reported a case of graphic aphasia in association with epilepsy in a case of cerebral tumor lasting nine years. The man's speech in this case was slow and paraphasic; while he was able to name most articles, some with which he was less familiar than with others, he could not name. He, therefore, had partial optic aphasia or word-dumbness. He could not write as well as formerly; this appeared to be due to the fact that he could not, except with difficulty, make out words on the right side of the line. He understood what he read; he wrote slowly from dictation and very inaccurately, few words of more than three letters were spelled correctly; the letters were badly formed. He copied from print to long-hand very slowly. In spontaneously writing he began well, but his efforts became worse and worse and in a half hour he could not write more than ten lines.

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[68] Barnes, Review of Neurology and Psychiatry, vol. i. 1903, p. 531.

The temporal field of the right eye was markedly contracted for all colors. In the left eye the nasal field was contracted about ten degrees.

The patient grew progressively worse until his death. Many symptoms and conditions not especially relating to the visual fields are omitted.

Necropsy showed a tumor about the size of a small orange, occupying portions of the postcentral, superparietal and inferior parietal convolutions, extending into the white matter but not as far as the ventricles. The growth was a gliosarcoma.

Admitting the existence of a cheiro-graphic or cheiro-kinæsthetic centre distinct from all other special centres, sensory or motor, Barnes argues from his case that the connections between the visual centres and both the graphic and motor speech centres, would be severely injured, this being accompanied by partial destruction of the visuo-auditory and cheiro-auditory commissures. It will be seen from Barnes' explanation that all forms of writing would be much impaired, while other aphasic symptoms were referable to partial injury of visuo-auditory commissures. His partial right homonymous hemianopsia was evidently due to lesion of the optic radiations.

Barnes accompanies his paper with an interesting illustration which is reproduced in Fig. 19.

Gulbenk[69] has described the case of a patient with a rare disturbance of the faculty of writing, which he terms *dysantigraphia*. A man, seventy years old, came into the hospital for difficulty with his speech and inferior facial paralysis, which had been present for about twenty days. Speech was slow and embarrassed, the words being badly pronounced but intelligible; syllables were not transposed and jargon-aphasia was absent. He had no paralysis of the limbs.

The patient could write pages fluently and legibly to dictation; but if asked to copy, he could only do one or two lines and then would begin to misspell and the writing would become illegible about the fourth line. If again dictated to, he could immediately write without fatigue.

The aphasia, of which a trace was present on his admission, soon developed more and more, becoming almost complete. At this stage writing was quite impossible.

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[69] Gulbenk. Rev. Neurol., February 15, 1904. Abst. in Review of Neurology and Psychiatry, vol. ii. 1904, p. 322.

Gulbenk holds that this case shows that the truth lies between the view that our hand copies visual images stored up in the angular gyre, and the other that our hand translates directly into graphic signs the sounds furnished by internal speech. In writing we use at the same time visual images and these sounds. Admitting a graphic centre at the posterior extremity of the second frontal convolution, Gulbenk explains the symptoms of the case by postulating a functional ischemia, leaving intact the connection of auditory (temporal lobe) with motor images, but interfering with the connection of visual images (angular gyre) with the same. This ischemia would affect the connecting fibres in their passage beneath the Rolandic convolutions, thus causing the paresis of the inferior facial. The author compares *dysantigraphia* with the dyslexia of Bruns, where the patient, after reading fluently a few words, finds it impossible to continue.

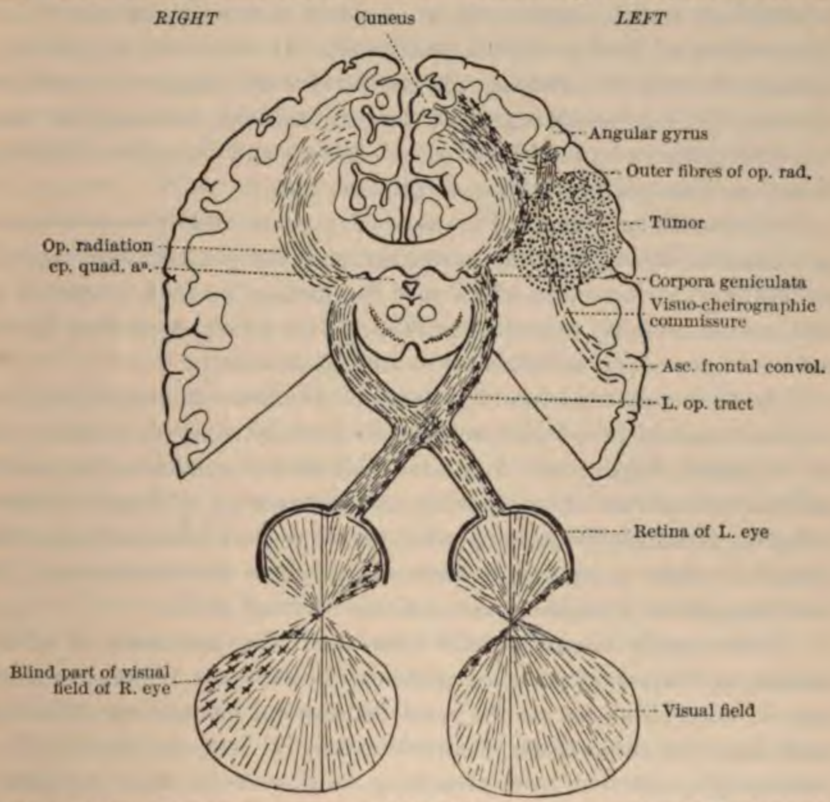

FIG. 19.—Diagram showing the manner in which writing and speech were impaired by destructive lesions involving the tracts between the visual and the cheiro-graphic and motor speech centres, and also involving the visuo-auditory and cheiro-auditory commissures. (Stanley Barnes.)

Numeral-blindness without either letter- or word-blindness is occasionally recorded; it would, perhaps, be more frequently observed if the attention of the examiner was directed to the subject. Hinshelwood reports four cases of word-blindness in only one of which blindness for figures or numbers was present. In one of these cases the patient, a teacher of languages, became suddenly totally word-blind and letter-blind, and yet, when he was examined with the test-types, while he could not read a single word, he could at once recognize and repeat the numbers at the top of each paragraph. In this case, the lesion, a considerable area of softening, did not involve the angular convolution, although it did the white matter of the occipital lobe, including the tract from the primary to the higher visual centres and the optic radiations as well as some portions of the occipital cortex.

Dejerine[70] has recorded a case of pure word-blindness without agraphia, due to a subcortical lesion, agraphia being added with the subsequent cortical lesion, in which case the patient was totally unable to read, and eventually, to write; he retained the power of reading figures and could do mental calculations as well as formerly.

I have seen cases of word-blindness in which the power to recognize and use numbers in calculations was retained in whole or in part. In my diagrams, Figs. 3 and 5, I have indicated a number-seeing centre as distinct from that of word-seeing and letter-seeing, although adjacent to both. Hinshelwood accords with the view thus indicated, that the groups of visual memories of letters and figures are deposited in distinct but probably adjacent areas of the cerebral cortex.

A few words might be said here about the treatment of visual aphasia, and especially of word- and letter-blindness. As many of these cases of word-blindness are the result of cerebral hemorrhage, Hinshelwood urges the importance of complete cerebral rest, and above all, of preventing the patient from practising reading for at least a considerable time after the attack. Many of these cases are the result of syphilis, and it is wise to put the patient on specific treatment if there is the slightest possibility of syphilis having been contracted. When actual destruction of the centre for word-seeing has occurred, re-education may call the corresponding centre of the right cerebral hemisphere into function, but re-education should not be attempted until all signs of acute brain disease have disappeared.

The treatment of alexia, due to word- and letter-blindness is sometimes disheartening, but with perseverance much can be accomplished, at least with persons of intelligence and education. Of course, as auditory, and to some extent, other forms of aphasia, are commonly associated with the visual speech disorder, training for word-deafness, paraphasia and partial motor aphasia, may need to come hand-in-hand with that for word- or letter-blindness, but I shall speak here only or chiefly of direct visual training.

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[70] Dejerine, Comptes-Rendus des Séances de la Société de Biologie, March, 1892, p. 64.

The patient should be made to copy numbers, letters and words, separately and in various juxtapositions. Efforts should also be made, as time progresses, to have him write from dictation and spontaneously. After writing from dictation or copying, the patient should try to write the same numbers, letters, words or combinations by recalling them for this purpose. With regard to words, it is probably best to begin with nouns, following with verbs, and later with qualifying and conjoining words, which are best taught by presenting them in short sentences.

Reference to the plan pursued in a recent case, the one recorded by Dr. Weisenburg and the writer, will perhaps give the best idea of the methods of training of most value for visual aphasia.

Two methods are used at the present time in teaching children the recognition of words. In our Philadelphia public schools, the word system is used,—that is, the child is not taught to spell the word, but to recognize it *en bloc.* An apple is shown or is drawn on the board. The child is told that it is an apple, and after this is firmly impressed, the word *apple* is written on the board, and an attempt is made to fix the image of the word in the child's mind. It is claimed for this method that by it words are acquired much faster. The child later learns of himself to apply the method of spelling, or, what is by no means unusual, never becomes a good speller.

In the older method the child is first taught the alphabet, and then to spell the word letter by letter, and syllable by syllable. While this method is a slower one, the results are better as regards spelling. It is probable that a carefully considered combination of the two methods would work out the best results.

Löwenfeld[71] records two cases of amnesic aphasia in which the patient, in one case, could not read short but could read long words; in the other case, if the patient had difficulty in reading a word, instead of trying to spell it, he would attempt to pronounce it *en bloc.* This was the case with the patient treated by Dr. Weisenburg and the writer. He could at times recognize words, and often when he would try to pronounce a word he would not attempt to either spell it or pronounce it in part, but as a whole. On being asked to recall a word with which he was familiar, his mental picture of the word was of its entirety; he could not recall its literal components nor spell it. In training him, both the word image and the letters method were used. In six weeks he was able to recognize nearly all the letters of the alphabet. Considering that he was only able to recognize about six letters when he came under our care, the improvement was marked. The letters were impressed in different ways. He was shown a letter, as *p*, made to pronounce it, to write it, to pick it out of other letters, and to try to picture it. At the same time, simple words were taught him in the same manner; in other words, he was shown the word, made to pronounce it, to write it, to pick it out of other words, and try to recall its appearance mentally. The progress in the recognition of letters and words was about equal.

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[71] Löwenfeld, Ueber zwei Fälle von amnestischer Aphasie. Deutsche Zeitschr. f. Nervenheilk., vol. ii. 1892.

AGRAMMATISM.

One who has many opportunities to study aphasics soon becomes aware that in those cases in which language is not entirely lost or is confined to a few simple recurring utterances, like "Yes" and "No" or "La la," "Oh no," or some profane ejaculation like "Damn" or "Oh God," the chief disturbance of speech exhibits itself in a disorder of grammatical expression. The patient, with his mutilated speech, tends to recur to the forms of expression which are used by those who, like the Chinese, have a language which is undeveloped so far as grammatical construction is concerned, or like the imbecile, the demented, or the victims of confusional insanity. Various parts of speech which are necessary to the full expression of the language ordinarily employed by the educated, or even by those who are largely uneducated, are omitted. The words used are transposed or emitted in grotesque confusion. The grammar of language has fallen into confusion or chaos. When this disorder of speech is the result of disease or defect, it is termed agrammatism or akataphasia, a subject which has received large attention from a few German and other writers, like Steinthal, Kussmaul, and Pick. Kussmaul has devoted an interesting chapter to these syntactical disturbances of speech. He shows that a speaker, in order to express himself correctly in language, must have an unbroken flow of words, perfect grammatical diction, and a correct arrangement of words. One or two, or all, of these requirements may be impaired or lost.

In the production of agrammatism or akataphasia it is evident that one must take into consideration not only the destruction of centres and of tracts, but also the more or less general disturbance of the entire zone of speech as it has been defined and described in this chapter. According to the position and extent of a lesion, the grammatical or syntactical derangement may be of this or of that kind. When auditory speech centres or their incoming, outgoing, or associating tracts are the seat of disease, the grammatical as well as the philological disturbance of speech will be dominated by losses or impairments which show that auditory language has been the subject of the most severe and destructive assault; when visual centres and tracts are the seat of lesions, visual language will suffer the most in grammatical as in ordinary graphic expression; and in like manner, when the anatomical substrata of the sphere of language on its motor or emissive side has borne the brunt of disease or injury, the disarrangement will be most shown in the power of conception and of expression. In each of these cases grammatical construction will suffer to a greater or less extent.

According to some authorities, these grammatical and syntactical disturbances of speech, or some of them, are due to the disease or defect in speech centres concerned with the co-ordination of languages; according to others, the morbid phenomena are the result of the dissociation of the separate centres and areas concerned with language. Probably both views have some foundation in fact.

As I have elsewhere pointed out,[72] the recognition of the part played by the impairment or loss of the power of grammatical construction or arrangement must receive special consideration in attempts at the restoration of speech by training. A citation from this paper on the Treatment of Aphasia may be of service in this connection.

"It became a matter of interest to me in observing aphasics who were striving to recover their lost powers to note the particular parts of speech which were most obscured and most difficult to regain, and also the particular difficulties which attended their reacquirement or prevented this. A patient who has so nearly lost language that he has only one or two recurring utterances regains, by repetition and recall, word after word, especially nouns. Later a few verbs are acquired. Adjectives, adverbs, prepositions, and articles, the parts of speech whose uses are to modify, limit, or express relation, are so entirely lost to the patient as to cause more than anything else his difficulty in language. The same remark applies to conjunctions like *and* and *but*, and with a special force to the auxiliary verbs in all their moods and tenses.

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[72] Mills, The Treatment of Aphasia by Training, Journal of the American Medical Association, December 24, 1904.

The meaning and use of such words or combination of words as *is* and *was* and *has been* are in eclipse or lost to a varying extent. The impairment or loss of the faculty of constructing sentences or propositionizing in language is one of the most important if not the fundamental defect. While thought is at times expressed by a single word, the sentence is the usual unit of thought expression, and the aphasic patient, or at least some aphasic patients, find their chief difficulty in regaining those parts of speech which are concerned with qualifying and correlating. The grammar of language no longer exists for them.

"Considerations of this kind led me to the use of primers and grammars in retraining of aphasics, and these with individuals previously reasonably educated are of great value. Patients should be taught the grammar as a child is instructed,—in other words, by teaching him the meaning of the different parts of speech and the exact methods of using them in phrases and sentences. The significance and value of the qualifying, relating and conjoining parts of speech should be enforced by numerous examples. When the aphasic, in his efforts to repeat after another or to respond to what another asks or indicates, uses incomplete sentences, the complete sentences should be given and explained. In every possible way the manner of using adjectives, prepositions, conjunctions, and auxiliaries should be impressed. The patient, with the book before him, should be taught to conjugate verbs, decline nouns and pronouns, compare adjectives, and in other ways go through the routine methods of studying language employed in the schools, these being modified by the particular requirements of the case. The dictionary can often be used as a valuable adjunct to the grammar, some patients taking a particular interest not only in reacquiring words in this way, but in thus learning their meaning and their uses as parts of speech."

Close examination of the subject, especially in connection with the study of actual cases, shows that the faults in language arrangement from which these patients suffer are, in a strict sense, syntactical rather than grammatical, and the term akataphasia is probably more suitable than that of agrammatism. Syntax is "the due form and arrangement of words or members of sentences in their mutual relations according to established usage. Syntax includes the proper use of parts of speech and of forms in their combinations to make sentences and their proper arrangement or collocation." (Century Dictionary.) A consideration of the numerous examples furnished by partial aphasics, or by aphasics recovering from various forms of disturbance of speech due to cerebral disease, shows that their great impairment is of the faculty of constructing sentences,—that is, of arranging the words of a sentence in their proper position and order. Grammar is a more general term, including syntax; but grammatical mistakes as they are commonly understood refer to the misuse of parts of speech which may be arranged in a sentence correctly from the syntactical point of view. It is a curious observation that aphasics, in the process of re-education, usually employ the parts of speech correctly or incorrectly according to their previous education,—that is, in so far as they regain their linguistic powers. Their sentences may be mutilated. Nouns may be present in considerable number, and they may employ some verbs and some qualifying and conjoining words; but the verbs used, if the patient is educated, are of the right mood and tense, and the substantives and pronouns are of the correct person and number. They do not use the plural for the singular or the present for the past. The uneducated, however, in proportion to their degree of education or their lack of it previous to the onset of their aphasia, make grammatical mistakes. As speech is reacquired, the professor or Jimmie Fadden, whatever may be his syntactical reacquirements, reasserts himself grammatically each after his own kind.

Pick,[73] in an article on agrammatism as the result of a focal lesion, gives several theories which have been held regarding this symptom. He believes that it is a so-called disorder due to lesion somewhere in the cerebral cortex, and holds that this lesion is situated in the left temporal lobe adjoining Broca's area. The case reported seems to prove this contention, for, although two areas of softening were present in the left hemicerebrum, one in the anterior part of the left supratemporal convolution and the other in the left third frontal convolution, Pick gives reasons for the opinion that the lesion in the anterior portion of the first temporal gyre was first in order of development and gave rise to agrammatism. Dejerine and his school, as is well known, hold to the sufficiency of Broca's area for all the purposes of motor speech and writing, and contend that agrammatism, like aphasia and agraphia, is only one of the varieties of a comprehensive motor aphasia. Bernheim [74] has adopted the views of Dejerine, but, as shown by Pick, an important study of the cases reported by Bernheim will show that the lesion in these cases was in every case in the temporal lobe, or at least involved this lobe.

Pick discusses the relations of grammatical expression to a cerebral substratum. Ziehen, for instance, holds that grammatical expression is not dependent upon the integrity of a co-ordinating speech centre, but is rather the result of associative combinations. Pick, however, holds to the existence of a language co-ordinating or grammatical centre which, as has been indicated, he places in the anterior part of the temporal lobe.

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[73] Pick, Zeitschrift f. Heilk., vol. xxiii. No. ii. 1902.

[74] Bernheim, De l' Aphasie motrice, 1901, Ausdruck verleihen.

VISUAL ANOMIA OR WORD-DUMBNESS.

One of the most interesting of aphasic clinical phenomena is that which is illustrated by the inability of a patient having good vision for ordinary purposes, suffering from a lesion in the cerebral zone of language, to name by sight an object the use and nature of which he recognizes. For this symptom, which has long been observed by clinicians, Freund,[75] who made the earliest elaborate study of the subject, suggested the name visual aphasia. Another term frequently used, especially by the Germans, is optic aphasia. Merkens[76] proposed to call the symptom word-dumbness, a term which has much descriptive appropriateness. It is, as Langdon[77] suggests, name memory-loss or anomia, or rather visual anomia.

The preferable names are visual anomia or word-dumbness, although the term optic aphasia has probably had the largest use. The expression visual aphasia employed by Freund is objectionable, as it may lead to a misunderstanding. I prefer to use this name rather to designate the entire class of visual aphasic phenomena, and in that sense it has already been used in the present chapter as a general caption for all the visual affections of language which have an organic cerebral substratum.

Occasionally word-dumbness is observed as an isolated form of anomia,—that is, it is only through sight that the inability to name known objects is exhibited; frequently the patient is unable to name objects which he recognizes through the sense of sight, and is also unable to denominate them through other senses,—as, for instance, that of touch or of hearing. The name memory-loss, in other words, may be not only for one but for several or all of the special senses. In one of my cases the names of objects could not be recalled either through sight or touch, but could by means of hearing. A gustatory or olfactory aphasia is possible, the patient being unable to name a rose, for example, through the sense of smell,—vision and the other senses being excluded,—or an apple through the sense of taste, with the same sensory exclusions. In still other cases the patient may be unable to name an object with the help of any senses, although he recognizes the nature and use of the object through all the senses.

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[75] Freund, Archiv für Psychiatrie und Nervenkrankheiten, vol. xx. 1888, p. 404.

[76] Merkens, Deutsche Zeitschrift f. Chirurgie, vol. lx., p. 417, 1900.

[77] Langdon, The Aphasias and their Medico-Legal Relations. The Laning Printing Co., Norwalk, Ohio, 1898.

In order to place clearly before the reader the idea of general anomia or loss of naming power through any of the senses, a well-recorded case by Potts[78] is worthy of citation. A man who was left-handed for all acts except writing lost the power of naming objects, whether they were seen, heard, smelled, felt, or tasted. He comprehended all that was said to him, knew the names of objects, and could converse fluently with the exception that he omitted the names of persons, places, and objects. He could not repeat names pronounced in his hearing, but he knew his own name and address and could write them; but otherwise spontaneous writing was impossible. He copied fairly well but soon tired. He had had an apoplectic attack, the immediate symptoms of which, with the exception of the visual aphasia, had disappeared. In about two months he made such marked improvement that he was able to name after reflection most objects presented to him, and he was also able to write a letter. This man, although word-blind, could read numerals. Hemianopsia was not present. Potts argued that it was necessary to assume in this case either that the lesion cut a number of tracts leading respectively from the centre of visual, tactile, auditory, olfactory, and gustatory memories to the motor centre, or else that tracts from each of these centres converged to a common centre where the memories of names are stored up, to be called into consciousness by stimulation of one or all of the percipient centres, and that from this centre a tract passes to the motor speech centre and that this tract was destroyed.

Personal experience with a number of cases with necropsies has demonstrated for the writer the fact that this symptom is present in lesions variously situated in the cerebral zone of speech, as the hinder part of the left third frontal convolution and the insula, in the third and fourth temporal convolutions, including both cortex and subcortex, at the junction of the posterior thirds of the first and second temporal convolutions, and again in these convolutions and the adjacent portions of the occipital lobe. Cases have been recorded which indicate that the symptom may arise from lesions situated in various portions of the occipital cortex or subcortex. Freund, after recording several cases of his own and reviewing the literature of the subject, explains the mechanism of the occurrence of the symptom with the assistance of a diagram.

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[78] Potts, Journal of the American Medical Association, May 4, 1901.

Bearing in mind the facts already presented regarding the centres for object-seeing and the various tracts and centres of the cerebral zone of speech, the production of the symptom from differently located focal lesions can readily be understood.

It is first necessary to recall the writer's views regarding a naming centre. I have long held that between the sensory and motor cerebral centres for speech exists a concept area, in which probably is a naming centre. This has been located on theoretical grounds and as the result of a few clinicopathological observations in the midtemporal region. Presuming the existence of this centre, a centre concerned with the affixing of names to concrete ideas of objects recognized through the lower and higher centres for the special senses, it is evident that word-dumbness will occur as the result of destructive lesions variously situated.

It has been shown that in the lateral occipito-parietal and occipito-temporal regions is situated an area concerned with object recognition; also that destruction of the whole or a part of this area, in some cases on one side of the brain and in others on both sides of the brain, causes the symptom which is known as object-blindness or mind-blindness. Of course, if the patient cannot recognize objects he will not be able to name them. The symptom, therefore, may result from a lesion of the region or regions of object recognition. Word-, letter-, and number-blindness, for instance, are due to a destructive lesion of the angulo-occipital region or the tracts leading to it, and inability to name words, letters, or numbers will be present as a part of these forms of cortical and precortical visual aphasia. When objects other than words, letters, and numbers cannot be recognized, it is probable that a bilateral lesion is usually necessary for the production of not only mind-blindness, but of visual anomia. Tracts pass from the various centres for object seeing to the naming centre in the left temporal lobe. A lesion of these tracts in the left hemisphere will cause word-dumbness for those objects whose recognition is confined to this hemisphere. For objects recognized by both occipital lobes it will be necessary for the complete production of this symptom that the tracts passing from both hemispheres to the naming centre shall be destroyed. This may be brought about by a single lesion after the tracts unite, by bilateral lesions, one in the left and the other in the right occipital lobe, or one in the left hemisphere and the other in the splenium of the callosum, through which it is probable that the tract passes from the right to the left side.

Lesion of the naming centre itself will also cause this symptom, and in this case it is probable that the word-dumbness will be not only for objects recognized by sight, but for those recognized by the other senses, as the tracts from all the centres of the special senses probably converge or concentrate in the concept naming area. A case like that reported by Potts would, for instance, be most likely due to a lesion situated here.

From the naming centre a tract also passes to Broca's centre, and destruction of this tract will produce word-dumbness. It will also be caused by lesion of the centre for word hearing, as the auditory verification of words used will not be possible. Certainly the symptom will result from lesion of Broca's centre, to which all tracts concerned with speech on its sensory and concept sides pass. It is scarcely necessary to say that anomia will be present if a lesion destroys the tracts from the retina to the basal and cortical centres for sight. Lateral hemianopsia is frequently combined with word-dumbness, because of the juxtaposition of the optic radiations to the tracts and centres which are concerned with object recognition and the processes associated with it. Destruction of the macular tracts and centres will, of course, interfere with the recognition and the naming of words, letters, and other objects.

CHAPTER IV.

GENERAL EXAMINATION OF THE EXTERIOR OF THE EYE AND THE REGION OF THE EYE; PERIMETRY; COLOR-BLINDNESS; PERIPHERAL BLINDNESS, AND SUBJECTIVE VISUAL SENSATIONS; AMBLYOPIA.

By CASEY A. WOOD, M.D.

Inspection of the Eye.—Much valuable information concerning the condition of the eye, not only of its external but of its internal structures, may be obtained by *simple inspection*. This is especially true of an examination made for neurologic purposes; there are many valuable signs of involvement of the nervous apparatus exhibited by the external eye that may easily be detected if the observer will take a little trouble. Such an examination must, of course, be conducted at close range and may be aided by instruments. To be effective it should be methodical and a certain order ought to be preserved in all cases. Great gentleness should be observed and as little handling of the lids and globes indulged in as possible, lest eyes already sensitive be rendered so irritable that further examination of them is rendered difficult, painful or impossible.

This **objective examination** of the eye should begin by seating the patient in a chair facing a window admitting indirect or diffused sunlight. The surgeon should then carefully inspect the superficial structures and parts surrounding the globe. The *lids* should first be examined closed. Do their edges approximate readily, entirely, and without apparent effort on the part of the patient? Does the dermal furrow describe its natural semicircle from one canthus to the other? Can the patient shut both eyes tightly, and does the wrinkled skin present about the same appearance on both sides? Is he able to open both eyes to their fullest extent and in doing so is one eyebrow drawn upward as high as the other? Is there contraction, tonic or clonic, of the orbicular muscle? On again assuming the primary position is there drooping of either lid? The lids in normal eyes should be in close apposition to the globe, and the small *puncta lachrymalia* should remain in intimate contact with the eyeball in almost all excursions of the latter. The margin of the upper lid should rest on the cornea, and this relation should be preserved in the downward movement of the ball; in other words, there should be no exposure of the white sclera between the upper lid-edge and the cornea proper.

Incidentally, one may examine the lids for irregularity of their outline, for œdema, distended vessels, redness, tumors, etc., and for crusts, discharge or ulceration along their margins and intermarginal spaces.

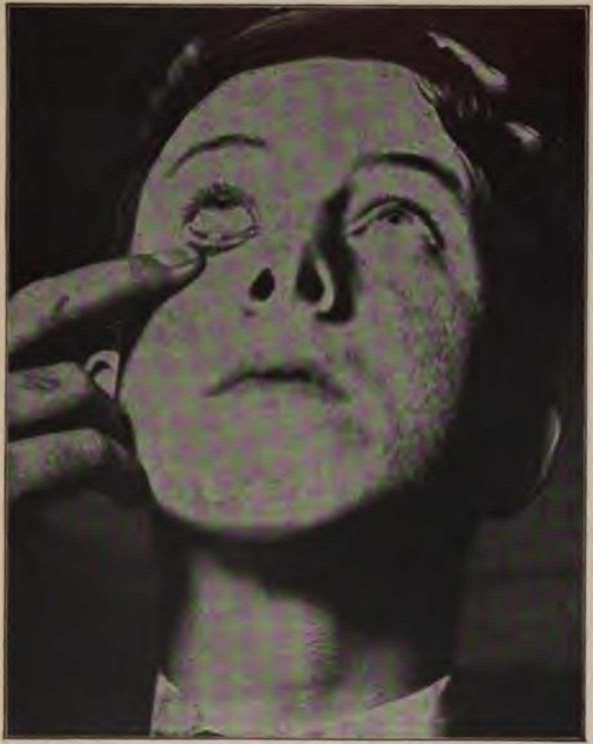

FIG. 1.—Everting the lower lid. (From Wood and Woodruff.)

The eyelashes also should be inspected, as to whether they are lacking, are abnormally numerous, misplaced, incurved, etc.

The position and sufficiency of the *lachrymal drainage* should be determined. If there be an overflow of tears its meaning should be decided; or as sometimes happens, to a paresis of the nerve-supply to the orbicularis; whether it be due to obstruction in some part of the lachrymal passages.

Both *lids should be everted* and the condition of the conjunctivæ, ocular and palpebral, determined. To do this thoroughly, and without hurting the patient, the tip of the thumb should be placed on the *lower lid* near its margin and the patient told to roll the eye upward. As he does this the lid is pressed backward, exposing the whole extent of the lining mucous membrane. Complete eversion *of the upper lid* is more difficult. The examiner stands behind the chair on which the patient sits, and the latter is directed to look down during the whole performance. The forefinger tip of the left hand is now placed upon the upper lid just below the eyebrow and the lid drawn by the finger up and away from the globe so that the lashes point upwards. The cilia and lid edge are now grasped by the forefinger and thumb of the right hand, care being observed not to touch or press upon the eyeball. The lid is now drawn steadily down and away from the globe while the tip of the left forefinger is again slipped to the upper margin of the tarsus and pressed *downward and backward upon the globe.* At this moment the lid may be readily turned back over the nail of the forefinger, exposing the upper sulcus with its covering of conjunctiva.

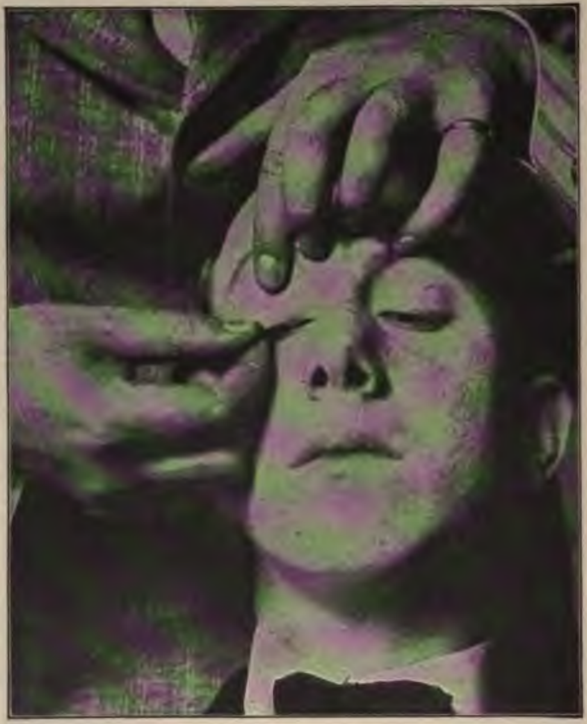

FIG. 2.—Everting the upper lid, first act. (From Wood and Woodruff.)

The position of the eyeball in the orbit can usually be determined at a glance, although when there is swelling of the lids and periorbital structures the relations of the globe to the orbital margins, or of one eye to the other, cannot always be determined in this way.

Proptosis, or abnormal protrusion of the eyeball, is noticeable only when one eye is more prominent than the other. It is usually by comparing one orbital region with the other that the degree of exophthalmus is determined. Where both eyes are unusually and equally prominent it is difficult to express such a proptosis in adequate terms. There are so many people who have physiologically prominent eyes that it is difficult to do more than express the degree of protrusion in relation to surrounding structures. Let the patient be placed in the primary position, when a straight edge, touching the cheek and supraorbital ridge, is placed over each globe parallel to and equidistant from the nasal crest. The distance between the front of the cornea and the straight-edge in each eye can be accurately stated in millimetres. In some instances, not necessarily pathologic, both cornea project beyond such a measuring stick, so that this simple test cannot be applied. In these cases the author has found the *ophthalmometer* of some value, for the purpose of determining whether one eye is the subject of a true exophthalmus or not. The patient's head being accurately and rigidly placed in the chin- and head-rest, the images of the mires are distinctly reflected from one cornea and the distance from the external canthus of the corresponding eye to the

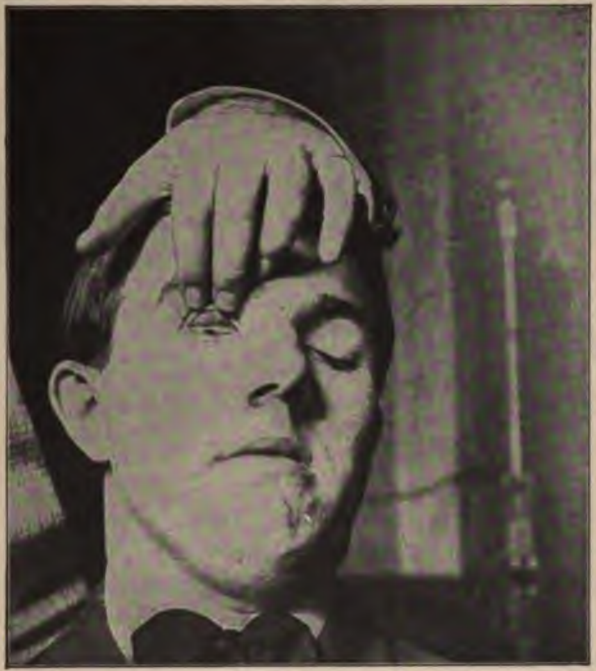

FIG. 3.—Everting the upper lid, second act. (From Wood and Woodruff.)

central point at the end of the telescope carefully measured. The observation is repeated on the other side. The difference between the two measurements expresses the amount of proptosis in the more prominent eye.

Various *proptometers* have been devised, but none of them furnishes more useful information than the methods just described.

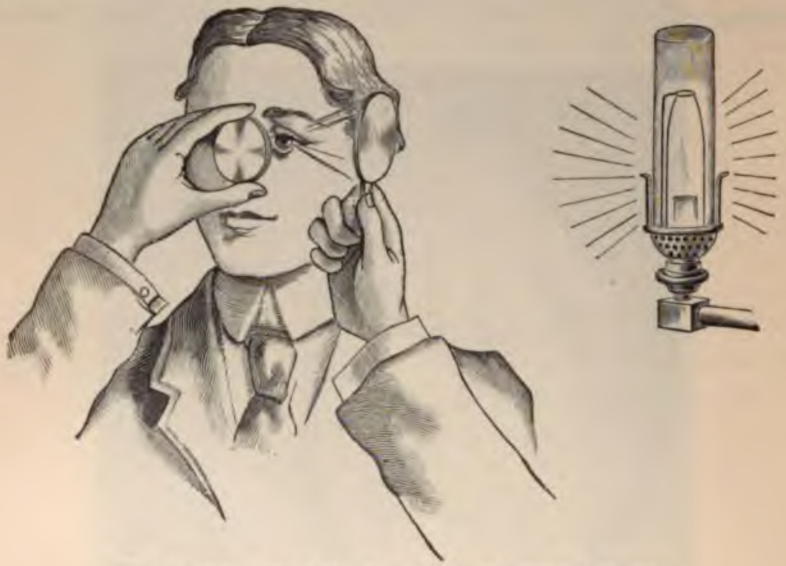

FIG. 4.—Oblique or focal illumination. (From Posey and Wright.)

Oblique Illumination.—The anterior segment of the eyeball is most satisfactorily and thoroughly explored by the aid of a convex lens, of about six centimetres focus, and a source of light in a darkened room. The rays from an argand burner or an electric lamp, placed about a metre to the side and a little to the front and above the patient's head, are allowed to fall on the eye looking straight in front. The lens is held so that the rays of light fall at right angles upon it and are focused *obliquely* on the eyeball. The eye is now moved in various directions so that the whole surface of the cornea and globe is illuminated by the pencil of light. In this way not only the external structures of the globe but the iris, anterior chamber, pupil, and lens are thoroughly illuminated and explored without producing any of the troublesome reflections that embarrass the observer when the rays fall upon the globe from a source of light situated *directly in front* of the patient. The value of this examination is enhanced by placing before the observer's eye another magnifying loop of the same focus, with which to inspect the area lighted with the aid of the other lens. This lens may be held in front of the observer's eye with one hand while the other directs the illuminating rays upon the corneal surface (Fig. 4).

FIELD OF VISION.

There is no examination of the ocular functions of greater value to the neurologist than the exploration of the extent and other characters of the visual field. Although the test is a subjective one and dependent somewhat upon the intelligence and coöperation of the patient it is, when undertaken with proper care and a due regard to the requirements of each case, difficult to overestimate the assistance it affords in diagnosis. The author entirely agrees with Wilbrand [1] that "*the measurement of the visual field* and the registration of the results in the form of a map have made it possible to represent graphically the intensity and extent of existing visual disturbances and to compare these pictures with one another. It can be proved that certain defects in the field of vision correspond with certain disease-processes. Moreover, a more precise diagnosis has been rendered possible by this method in lesions of those parts of the optic tract in which the ophthalmoscope cannot afford any assistance. Perimetry has obtained a still greater diagnostic importance, since careful study of the results of microscopical examination, with exact sketches of the field of vision, has widened our knowledge about the partial course of the fibres in the optic tracts, and seems about to fix the distinguishing symptoms under which neuritis, primary atrophy, and purely functional disturbances make their appearance in the visual field, as well as with regard to their prognosis and treatment."

On the other hand, it must be equally remembered that hasty and injudicious measurements of the visual field are likely to be valueless, misleading and harmful.

Various forms of the instrument (*perimeter*) used for determining the extent and character of peripheral vision have from time to time been devised, but, for all practical purposes, they may be divided into two classes, first, those intended for patients who can sit upright during the examination without much general or ocular fatigue; second, those by which the measurements are taken while the patient is in the prone position. The usual form of the perimeter employed in the first class of cases is that devised by McHardy, Landolt, Skeel, and others. The author prefers the first mentioned. It consists of a semicircular arc or band which revolves upon its centre so that it can be placed in the direction of any meridian. This arm is divided into degrees, beginning with 0 at the centre and extending to 90° at either extremity. At the centre of the semicircle is a small object (*fixation point*) for the eye to look at. The patient's head is supported by a chin-rest, one eye is covered, and the other, under examination, directed to look at the fixation point at the centre of the semicircle. A small white or colored object, varying from 5 to 20 millimetres square, is then carried from the periphery along the inner surface of the arc. The point at which it first comes into view in each meridian denotes the limit of peripheral vision for that meridian. These points (marked on a chart) when joined by lines give the boundary of the field of vision. If any defect is found during the examination the movable objects will appear blurred or disappear entirely. This defect is known as a *scotoma.*

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[1] Norris and Oliver, System of Diseases of the Eye, vol. ii. p. 191.

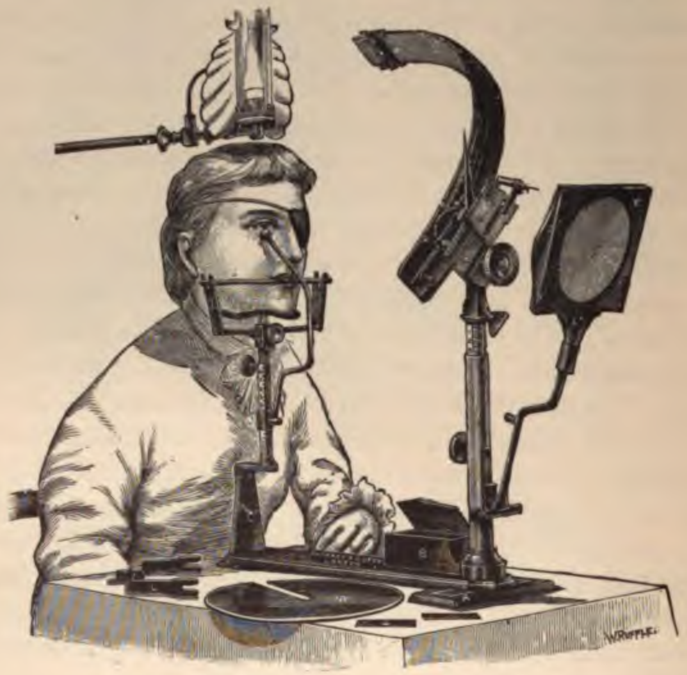

FIG. 5.—The McHardy Perimeter. (From Posey and Wright.)

A *physiological* scotoma is present in every field of vision. It corresponds to the optic nerve entrance and is situated about 10° to the outer side of the point of fixation, and is known as the blind spot of Mariotte. When a perimeter cannot be obtained the field of vision may be roughly measured by the hand in the following manner: the patient and the examiner face one another, the former with his back to the light. If the left eye is to be tested, the other eye is covered with a towel. The examiner closes his right eye and each looks into the other's eye at a distance of about a metre. The hand is then carried to the periphery of the field at an equal distance between the patient and himself. It is then moved slowly inward towards the visual axis and the patient is required to say when he sees the finger-tips. This is repeated in the various meridians of the field of vision. If the patient sees the fingers at the moment they are also seen by the examiner, his field of vision is normal. If the fingers are not seen until they are nearer the visual axis than when seen by the examiner, there is some defect in the patient's field of vision. The perimeter-arc and the test objects are best illuminated by ordinary, diffuse daylight from a window, to which the patient presents his back. When that is not obtainable gas or electric lamps may be employed, care being observed that the illumination is uniform in the various positions of the arm of the perimeter.

Myles Standish employs, as a rough test for absolute defects in the visual field, a small electric lamp and conducts the examination in a dark room. One eye of the patient is completely excluded by means of a folded towel held over the orbital region by the palm of the patient's hand while he fixes the observer's nose with the uncovered eye. An electric lamp, of the smallest size, is employed as the test-object instead of the finger-tips in the preceding test.

Sweet has devised a perimeter in which the objects are white and colored electric lamps, but the writer has not used it.

On every perimeter chart used for mapping out a field of vision, a note should be made roughly stating the kind and amount of illumination employed, such as " bright day," " dull daylight," " electric light," etc., as the size of the field is, in some instances, much affected thereby.

Another rough means of defining the limits of the field of vision is the ordinary school blackboard. Although theoretically insufficient it is a fairly good substitute for the perimeter. In such cases black rods with round or square tips, or pieces of chalk, white and colored, are employed as test objects. While the patient fixes a white chalk mark on the blackboard, these objects, moved about as on the arc of the perimeter, map out the visual field.

The *portable perimeter* of Wilbrand,[2] or one like it, is valuable in the examination of bedridden patients. It is used in the wards of a hospital or on occasions when the stationary instrument is not accessible.

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[2] Norris and Oliver, System of Diseases of the Eye, vol. ii. p. 210.

The inventor terms it a *bed-perimeter*. "A wooden board (*a*), the breadth of which is equal to the length of the perimeter, and the length of which equals the width of the bed, is made so that it can be raised or lowered like a reading desk. A second board (*b*), which can be placed at any degree of inclination upon two perforated irons (*c*), is supported by putting a little iron pin (*d*) into a series of holes. Upon the board, *b*, is placed the perimeter. The board, *b*, is connected with the board, *a*, on the side turned towards the patient, by means of two hinges, which are placed near the corners. Both boards between the hinges are hollowed out so as to fit the regions of the chest and abdomen. The perimeter itself is supported below by a clamp (*f*) ,which is fixed vertically to (*b*). The board (*a*) is supported by four wooden screws (*g*), which are fastened by pairs to a rather narrow board (*h*) resting horizontally on the lateral bed boards. On the board (*h*), near the outer end is placed a clamp (*k*), so as to prevent the whole apparatus from moving sideways. A head-rest is so arranged that the chin of the patient can be brought into a comfortable position on the chin-rest of the instrument."

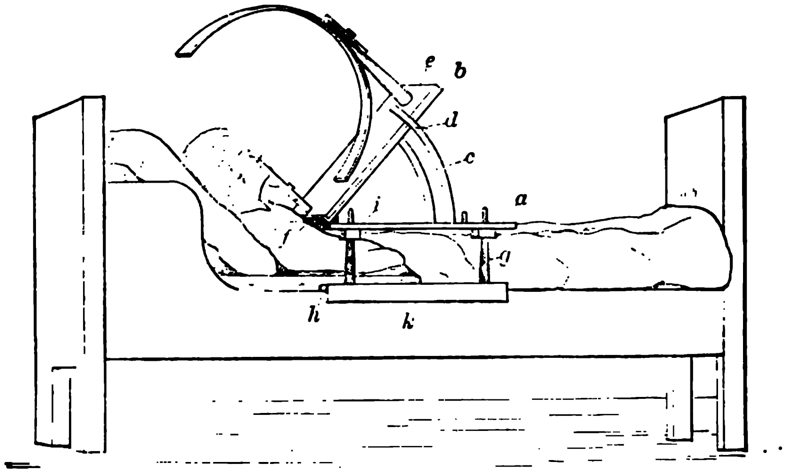


FIG. 6.—Wilbrand's bed-perimeter. (From Norris and Oliver.)

The best results in perimetry can be obtained only by the exercise of much patience and the expenditure of considerable time. Many neurologic patients after a short examination exhibit the so-called "retinal fatigue" and many others have such dull peripheral vision that the limits of their fields, especially for colors, are not easily defined. Again, it is not an easy task for either the examiner or the examined to locate and measure scotomata, particularly if they are small or incomplete. Frequent pauses should be made during the examination, so that the patient may rest his eyes, and if fatigue-signs are marked the completion of the examination should be deferred until another sitting. Unfortunately, it is in just such instances that control experiments should be made, that is to say, it is in cases exhibiting a " fatigue" neurosis that one must be most careful to repeat his observations and where it is desirable that there should be no " nerve-tire" produced by the efforts of the physician to conclude an examination. The patient should be encouraged to close his eyes, sit back in his chair, and rest himself as often as need be.

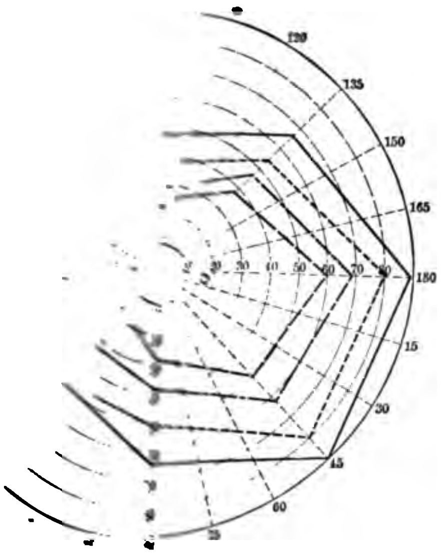
These difficulties are further increased when, for any reason, the patient is unable to see the fixation point on the perimeter. It is then advisable to have him place the tip of his forefinger over the point of fixation and, by virtue of his sense of locality, keep his eye in the primary position. The examiner must watch him closely to see that his gaze does not wander.

Conditions that Determine the Limits and Other Characters of the Visual Field.—It is particularly desirable to remember, in examining neurologic cases, that a number of factors enter into the results obtained by a perimeter and that many conditions may modify the shape and area of the field. Our standard should be the extent and boundaries of the visual field in the normal, emmetropic eye. Although, as before stated, the examination is essentially a *subjective* one we cannot always obtain the same results in every measurement even in the same patient or from observations of the same eye, but the error is generally so slight that it can be ignored, or at least borne in mind when considering the total value of the evidence presented by this and other means of diagnosis.

The **shape of the normal visual field** is an irregular oval, due to the fact that the retina extends farther forward on the nasal than on the temporal side, thus pushing the limits on the perimeter-map correspondingly outward.

The **boundaries** of the field of vision are also affected by the prominence of the eyeball in its socket, by the tip and bridge of the nose, the cheek and even by the size of the lips and moustache. They are often measurably widened by opening the eyes to the fullest extent, and there is no doubt but that, other things being equal, a larger field is found with a dilated pupil than with a narrow one. Owing to the long antero-posterior axis in *myopia* and the shorter optic axis in *hypermetropia* the field is smaller in the former and larger in the latter case. Bearing these facts in mind one should always have the patient under examination wear his correcting lenses and not hesitate to secure a full measurement

The inventor ... separate his lids of which is ... extent be overcome equals the ... during the exam- like a ... respectively. degree of ... character of the pig- putting ... amination, the nature is placed ... and with individual on the ... acute perception of are ... lowed eter



white; ------ blue; —-—-— red; ——— green.

Wright).

... men. For these reasons the charts ... considerable variations as to the extent ... as the largest area in the chart of the ... red, then green.

... the size of the field for white (taken ... little with the illumination, quite ... tests are used. This fact should ... for nervous disorders.

... field for white in *pathologic* states with ... are not infrequent. We see this espe- ... hemeralopia as encountered in retinitis ... choroiditis, high degrees of myopia, secondary ... and the like (See Chapter VIII).

Much has been written upon the need of standardized color-objects for the perimeter and, doubtless, greater scientific accuracy would be attained if these always exhibited the same color intensity or saturation. Colored test-objects should be as small as is compatible with distinct vision; in cases of amblyopia they may be as large as 20 millimetres square. It is well to remember that scotomata, especially of limited area, are best mapped out by the use of much smaller objects, whose sides measure only two or three millimetres.

The author has long been in the habit of using a single strand of white, red, green and blue wool for *detecting the presence of central color scotomata.* Double the thread between the thumb and finger of the right hand, holding it so that only a quarter of an inch of the loop projects and is seen. Patient and surgeon should be seated opposite one another. Cover the patient's *left* eye with a closely-fitting shade, and ask him to fix steadily the observer's *right* pupil at a distance of two-thirds of a metre. Interpose the small loop of wool in the common line of vision at a point a few centimetres in front of the examiner's eye. In relative central scotomata the patient will not recognize the color of the small loop (or it will appear less distinct) until it is removed a few millimetres to the right, left, up or down from the line of sight, and the distance it is necessary to carry the test loop in the various

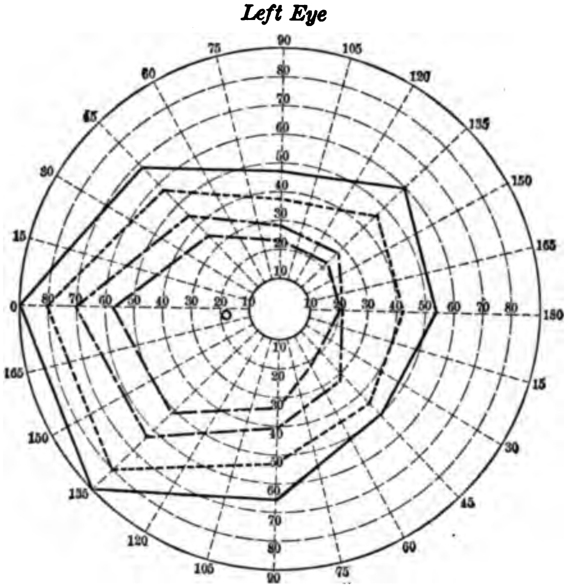
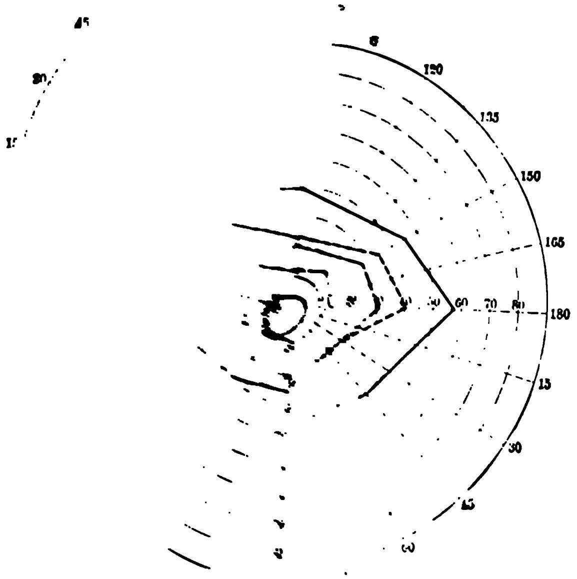


FIG. 8.—Normal field of vision, left eye. ——— white; ------ blue; —·—·— red; — — — green (From Posey and Wright.)

of the field by asking him to open his [illegible] idea of the size and with the fingers. The other difficulties [illegible] applied to the left by throwing the head first backward [illegible] charted by means ination of the vertical and horizon[illegible]

The extent of the field for colo[illegible] the manner in which ment used, the size of the obje[illegible] ays making perimetric of the background against w[illegible] closely resembling one peculiarities. For example [illegible] efinite information and [illegible] measurements will only [illegible] xample, it has long been



[illegible] and Wright.)

[illegible] lly, there is a zone of doubtful [illegible] boundary line of most fields. [illegible] observer and the intelligent patient [illegible] he sees it and then he doesn't see [illegible] repeated examinations are made; [illegible] changed the patient will within [illegible] area of [illegible] and white-per[illegible] To a slight extent practice [illegible] perceptive elements.

[illegible] object is moved from the centre to the [illegible] the opposite direction; or when the object is "wiggled," or kept in motion, about the zone of doubtful perception. Of course, it is wider when a large, shiny object is employed than when a dull, small one is used.

Central scotomata result from disease of the macular fibres. These are termed *absolute central scotomata* when all light perception is wanting. Defects for one or more colors only are called *relative* central scotomata. When white is seen as "grayish," or colors are appreciated only as tints of the test-object, the defects are known as *inaccurate* central scotomata. Finally, Forster adds the terms *positive* and *negative* central scotoma, defining the former to be that condition in which the patient reports a dark spot covering the fixation-object and the latter when the point of fixation appears blurred or indistinct. The precise conditions of the central field should always be indicated on the perimeter chart or noted on its margin.

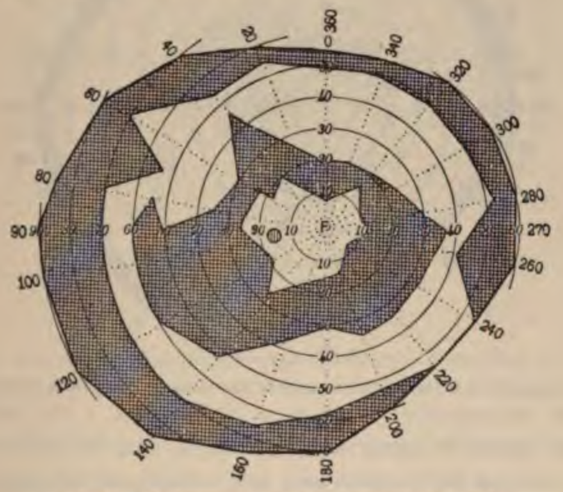


FIG. 10.—Ring scotoma. (From Norris and Oliver.)

The foregoing distinctions are now and then valuable in diagnosis, because negative central scotomata are generally the result of disease of the papillo-macular bundle of optic nerve-fibres, while positive defects are generally due to peripheral alterations in the macular region, often chorio-retinitic changes. Among the numerous forms of central scotoma an important one for the neurologist to remember is that due to "retinal fatigue," not infrequently seen in functional nervous diseases (See Page 108).

The *position* of central scotomata with reference to the point of fixation is indicated by the terms *paracentral*, when the defect lies mostly without the centre, and *pericentral*, when the fixation-point forms the centre of the defect.

Enlargement of the blind or Mariotte's spot must not be overlooked in searching for defects in the visual field. This may be due to congenital conditions, such as the presence of opaque nerve-fibres, but it is also one of the early signs of certain forms of optic atrophy, of chorio-retinitis of the fibres surrounding the nerve-head and it is seen in most cases of "choked disk."

Ring Scotoma.—The area lying between the fifteenth and forty-fifth parallels of latitude is called the *intermediate zone*. It is occasionally subject to fairly well defined defects, which, enclosing an otherwise normal central field of zone, constitute a "ring" scotoma (Fig. 10).

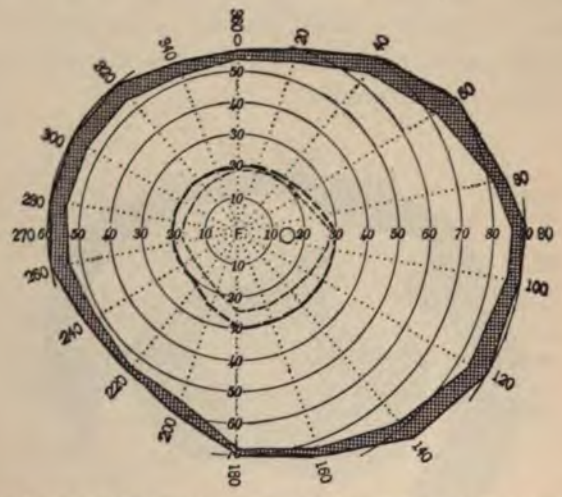


FIG. 11.—Defect confined to the peripheral zone of the visual field. (From Norris and Oliver.)

This annular defect is sometimes found, of varying width and shape, in syphilitic lesions of the chorioid and retina, in multiple sclerosis of the optic nerve and a few other conditions.

The peripheral zone of the visual field may be defined as that lying between the forty-fifth parallel and the extreme limits of the field. Wilbrand says that it generally begins in a normal eye where the perception of a five-millimeters blue object ceases. Isolated defects in this area do not disturb the vision of the patient to any extent, and we are mostly concerned with it in the diagnosis and prognosis of optic and other nervous affections. When the whole or most of the peripheral zone is insensitive to light patients realize the value of indirect peripheral vision. They act and feel as if they were seeing through a tube, and when the contraction extends still further towards the centre "telescopic vision" is the result, obliging the one so afflicted to look about him continually to obtain satisfactory sight (Fig. 11).

Hemiopia, or (better) Hemianopsia, is characterized by loss of one half or portions of half the field in each eye. It may result from disease in any part of one optic conducting path from the calcarine fissure to and including the chiasma (Figs. 13, 14). It is almost invariably *homonymous,—i.e.*, affects the optic radiations supplied to the two eyes from one side of the brain without involvement of either fixation-point. Binasal, bitemporal, or *heteronymous* hemianopsia is quite rare. When one considers for a moment the anatomic arrangement of the crossed and uncrossed fibres that constitute each optic nerve the explanation of homonymous hemianopsia is easy (See Chapter I).

Left Eye

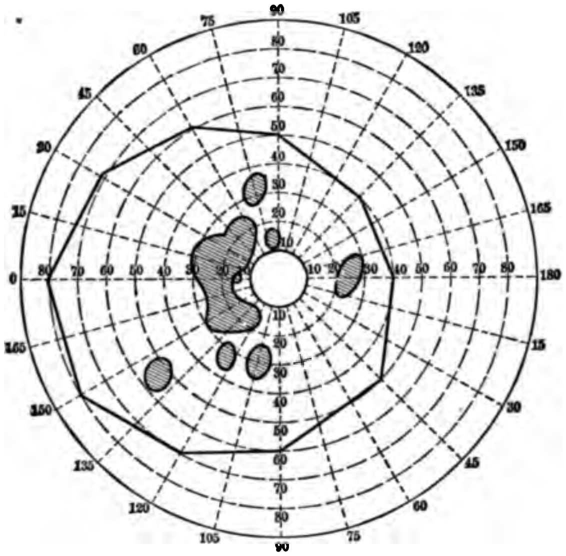


FIG. 12.—Scotomata as seen in disseminated chorioiditis. (From Posey and Wright.)

Of 154 cases of homonymous hemianopia collected by Wilbrand 74 were on the right side and 80 on the left. The line dividing the blind from the seeing half of the retina does not, as a rule, pass directly through the fixation-point. Fortunately, the functionating half usually includes the fixation-point,—*i.e.*, the lesion does not affect the fovea, because there are sufficient unaffected fibres from the normal visual centre supplied to each papillo-macular bundle to permit of good central vision. Inasmuch as the arrangement and distribution of the crossed and uncrossed bundles varies greatly in individuals the position of the frontier-line also varies accordingly. As a rule, this boundary runs in an almost perfectly vertical direction until it approaches within 5° to 10° of the fixation-point. It then diverges, curves around the central point and at the same distance below once more resumes its vertical direction. The functionating area about the point of fixation may have a considerable radius—from 3° to 10°, or even more.

The variations in the line dividing the seeing from the non-seeing retina correspond to variations in the size of the hemiopic fields, only, as a rule, the same part of the field is affected in each eye even if they vary much in extent. Later, part of the field may be recovered on one side while undergoing no change in the opposite eye, rendering the contrast between the two perimeter charts still more marked than before.

Right Eye

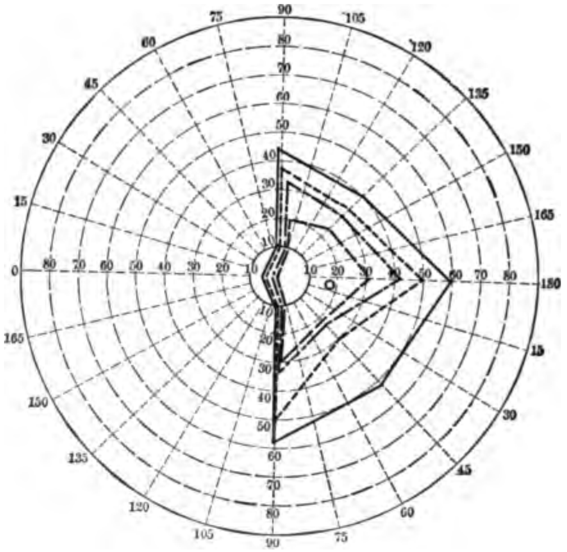


FIG. 13.—Field in left-sided hemianopsia. (From Posey and Wright.)

Incomplete hemianopsia is due, in the majority of instances, to destructive lesions of the occipital lobe. Defects confined to either the upper or the lower quadrants of the field indicate destruction of the lower or upper elements of the cuneus (Figs. 15, 16).

The periphery of the unaffected half of the field in hemiopia may or may not be contracted and the half-field is usually smaller on the side opposite the lesion (See Chapter IX, p. 391).

The field of vision in binocular fixation is, of course, larger than that of each eye taken separately. Although it is the field generally considered by the patient and ought, consequently, to be of particular interest to the neurologist, its practical value, apart from the subject of squint, is not very great.

Hemichromatopsia.—In true hemianopsia perception of color is lost with the field for white. In rare cases, however, there is a true ***hemiachromatopsia,*** in which the white field is unaffected (or white objects are seen as gray), the dividing line passing through the fixation-point. In explanation of this curious condition Wilbrand claims that there is a centre for colors distinct from that for form or from that for white. Gowers suggests that instances of hemichromatopsia would be more frequently noted if the color fields were tested at the bedside in all cases of cerebral disease (See Page 392).

An admirable summary of the *diagnostic values of defects in the visual field* is given by Gowers.[3] Loss of the pupillary reaction with

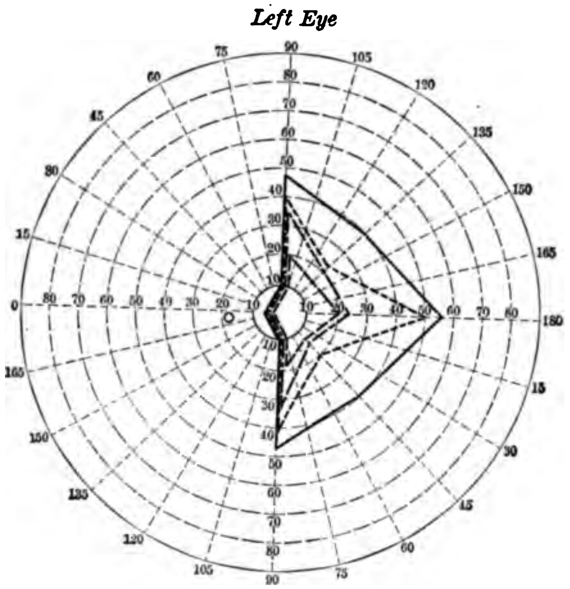


FIG. 14.—Field in left-sided hemianopsia. (From Posey and Wright.)

defective sight in one eye without affection of the opposite organ indicates disease of one optic nerve. A *central scotoma* usually means disease of the macular bundle of optic fibres, either in the body of the nerve or at the fovea itself. *Peripheral limitation of the field* may be due to (*a*) damage to the peripheral layer of the nerve or (*b*) a lesion of the whole nerve, which shows itself only in the periphery, where vision is normally duller than it is about the macular region. A *sector-like defect,* confined to the field of one eye indicates destruction of a portion of one nerve. An amblyopia, with concentric limitation of the field, marked in one eye but slight in the other, is due either to atrophy or to disease in one hemisphere. In the first instance the pupillary reaction is sluggish and the ophthalmoscope generally reveals the atrophic changes; in the second the nerve appears normal, the pupil acts promptly to light, the onset of the visual disturbance is sudden and there are usually other symptoms of cerebral involvement. In *functional, or hysterical, amblyopia* the symptoms are the same as in the second instance, but there are other hysterical stigmata present. In those rare cases of monocular hysteria the perfect reaction of the pupil serves to distinguish the functional affection from organic disease of the nerve.

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[3] Diseases of the Nervous System, vol. ii. p. 162.

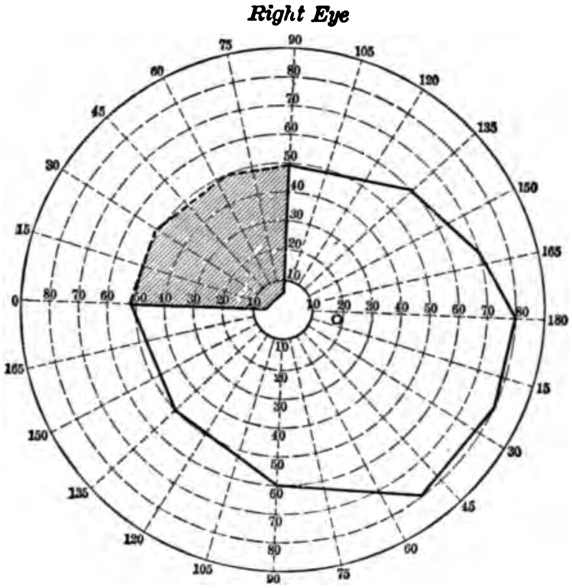


FIG. 15.—Homonymous quadrant hemianopsia. (Posey and Wright.)

Temporal hemianopsia means disease of the chiasma. Complete blindness of one eye with loss of the temporal field in the other generally points to disease of the chiasma extending to the outer fibres of the nerve or optic tract on the side on which there is complete loss of vision.

Wilbrand's or Wernicke's Light-test—the so-called "hemiopic pupil-reaction"—is useful in determining whether the lesion in a case of lateral hemianopsia lies in the *tractus opticus* or in the cerebral centres behind it (Fig. 17). The test depends upon the fact that when light is thrown upon the blind half of the retina the pupil contracts just as promptly as it does when it is projected upon the seeing half of the disease confined to the cortex or basal neurons. When the tract is involved —*i.e.*, when the conducting path to the corpora quadrigemina is diseased —it does not contract.

The patient should sit (as for an ophthalmoscopic examination) in a half-lighted room with one eye carefully covered and fixing with the eye to be tested an object directly in front, at least ten feet away. The rays from a small electric tube-lamp should be first focused on the forehead and then allowed to strike only the blind half of the retina. The effect is noted and compared with the result obtained by illuminating in the same way the other retinal half (See Page 393).

The **Æsthesiometer** is of considerable value in the examination of the eyes of neurologic patients. Variations in the tactile sensibility of the palpebral skin are best tested and mapped out by either Carroll's or Sieveking's instrument, but conjunctival and corneal areas of anæsthesia or hyperæsthesia cannot, for obvious reasons, be so measured. The author has found that a small camel's-hair brush answers every useful purpose. Employed with a little care, particularly where it is possible to compare a normal with an abnormal eye, valuable information can be obtained, not only in functional affections—mostly hysterical—but in those organic lesions—paretic and other—that reduce or increase the conjunctival and corneal sensibility. In examining the patient with this end in view, it is well to have him rotate the eye upward as much as possible, while very gently touching the lower conjunctival surfaces and lower half of the cornea with the tip of the brush. Then, the upper lid should be everted and the exposed conjunctivæ and upper hemisphere of

Left Eye

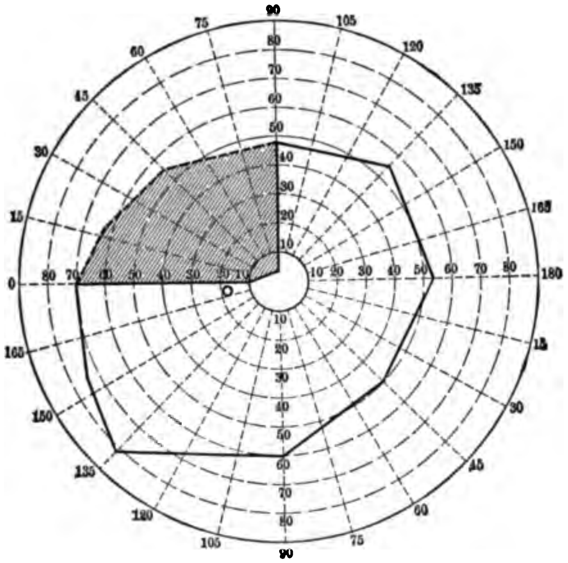


FIG. 16.—Homonymous quadrant hemianopsia. (Posey and Wright.)

the cornea are similarly tested. In this way the nervous patient does not *see* the approach of the brush-tip and is obliged to regulate his answers by touch-sensations only.

COLOR-BLINDNESS.

Color-blindness is either congenital or is due to some organic change in the optic nerve-fibres or the visual centres from which they spring. The latter class is best illustrated in toxic (tobacco or alcohol) amblyopia, optic atrophy, diabetes, hysteria, etc. Congenital anomalies of the color-sense, due to organic defects in some element of the optic radiations, are more common in men (four per cent.) than in women (one per cent.). (See Page 92). It is well to remember that while this congenital defect cannot be remedied and that the person born color-blind is always color-blind, the intelligent individual establishes a standard of color for himself and so frequently discounts some of the drawbacks that reside in a dyschromatopsia. Occupations like that of railroad fireman or engineer, requiring acute perception of colors, should not, of course, be undertaken by color-blind individuals.

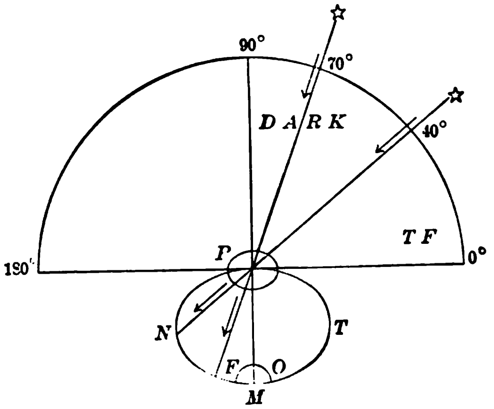
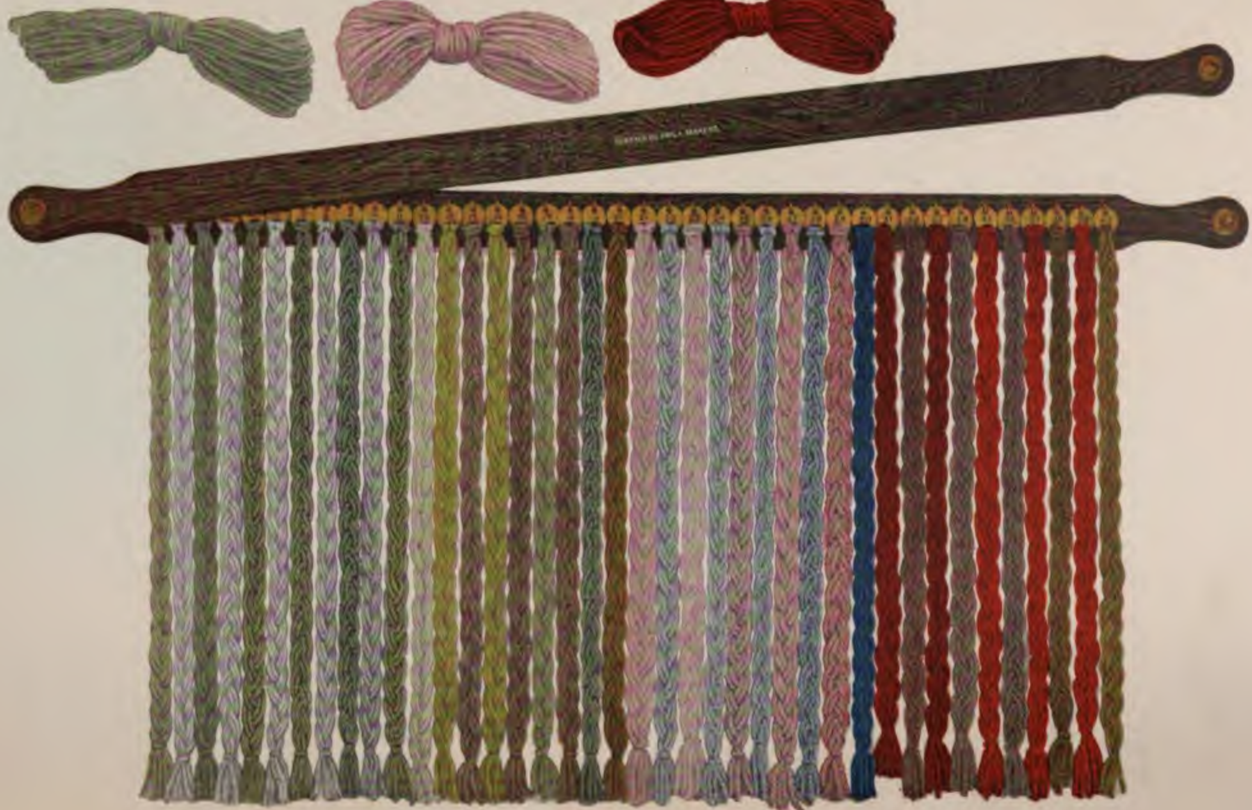


FIG. 17.—Illustrating the test for hemianopsic pupillary inaction; the lines represent a horizontal plane through the left eye and its visual field: *F O*, fundus oculi; *M*, macula lutea; *N*, nasal half of the field which is anæsthetic in temporal hemianopsia; *T*, temporal half of the retina; *T F*, temporal field; *P*, pupillary aperture, 180° to 0°, the equatorial arc or semicircle; 90°, vertical point and line passing through centre of eye to *M*; 70° and 40°, rays of light striking the insensitive nasal half of the retina, producing the pupillary reflex. (Seguin.)

Color-blindness is, for clinical purposes, readily detected by the use of various colored skeins of wool after the plan devised by Holmgren. Thomson's "color-stick" (Plate I) constitutes a handy arrangement of the skeins, which are numbered to correspond with a private key for the use of the color-blind examiner. The patient is seated before a le with, preferably, a sufficient natural illumination from a side

PLATE I.



Queen & Co., Phila.

Thomson's Color Stick.

window, on which are placed skeins of colored wools in different shades of red, green, gray, brown, etc. In front of him is a large skein or knot of pure green wool. This is the " test" skein, and he is required to select from the others in front of him the skeins of the *same color,* but not necessarily of the same shade. If there is any defect in his color vision the patient will select a number of the confusion colors (chiefly grays), with perhaps some green tints. If he accurately chooses all greens the investigation need proceed no further; he has normal color-sense—is not color-blind. If, however, he fails, he is given a rose-colored skein and again told to choose wools of that *color.* He is now likely to match the rose with browns, purples, dark grays or even greens. Finally, to find out what variety of color-blindness he has, he is given a deep red skein, to match which he will probably choose the dark browns and greens. The great majority of color-blind people are defective in red and green and, consequently, are said to have red-green-blindness, although all varieties, from simple green to total color-blindness, exist.

With Holmgren's test it is almost impossible for one who is color-blind to escape detection. Thomson's modification of this test is probably the most convenient. The person under examination should not be allowed to name the colors; only to select those colors resembling the test-skein in his hand.

Peripheral Blindness.—Generally speaking, blindness is regarded as that loss of sight that incapacitates the individual from following any occupation requiring the use of the eyes. The proportion of blindness due to peripheral causes—*i.e.*, those affecting the external organs of vision—is large. The tables of Magnus show that at least 67 per cent. of 2528 cases occurring in Germany were due to idiopathic eye affections. On the other hand as high as two per cent. resulted from purely cerebral diseases; injury, congenital defects and general affections being accountable for the remainder. Gonorrhœal infections of the conjunctiva (*ophthalmia neonatorum,* gonorrhœal ophthalmia of the adult), trachoma and diseases of the cornea induce blindness chiefly by producing scars in the clear corneal tissue. These infections were alone responsible for blindness in 27 per cent. of Magnus's cases. Spinal optic atrophy as well as irido-chorioiditis from meningitis (particularly the epidemic cerebrospinal variety) are common causes of lost vision.

When sight is lost from destruction of the cornea the necrosed tissue is replaced by cicatrices (leucomata especially) that are readily detected by the naked eye. More deeply seated opacities of the media, such as cataract, are better seen by the aid of the lens and the oblique illumination, already described on page 156. The use of the *ophthalmoscope* is required for the identification of many lesions of the posterior structures of the globe that with equal certainty produce blindness. Diseases of the chorioid, retina, and optic nerve, changes in the vitreous body, intraocular growths, glaucoma,—all these generally require an exploration of the posterior chamber and fundus oculi through the pupil for a positive diagnosis. The employment of the ophthalmoscope in the determination of sources of blindness is all the more imperative since the uveal tract, optic nerve, and retina constitute the anatomical seats of more than half the blindness one meets with.

SUBJECTIVE VISUAL SENSATIONS.

Peripheral Subjective Visual Sensations.—Muscæ Volitantes; Phosphenes.—The appearance of specks, spots, or other minute objects floating in front of the eye may or may not depend upon organic disease of that organ. In most instances muscæ are due to a hypersensitive percipient apparatus which takes notice of the normal opacities in the vitreous, but it is not uncommon to discover exudates and other floating deposits within the vitreous whose presence easily accounts for these appearances.

Phosphenes are colored rings or flashes of light which may at any time be induced by pressing on the inner aspect of the closed eye. These bright color sensations may also be caused by cerebral diseases, by optic nerve affections, by glaucoma, and by other conditions.

Hallucinations of Vision.[4]—Although in a restricted sense most phosphenes and muscæ volitantes are examples of visual hallucination they are not usually regarded as such. The sensations of this order due to cerebral diseases are on the other hand mostly genuine delusions, just as we know them to be in acute alcoholic mania. Hansell gives three varieties of visual hallucinations; first, those due to ocular disease, whether organic or peripheral; second, those that arise from localized disturbance of the cerebral circulation, including functional nervous affections, and, third, those resulting from organic disease of the cortical centres. Purely ocular hallucinations, as Hansell points out, are dependent upon a number of causes. These he enumerates as follows: (1) errors of refraction, (2) oculo-muscular defects, (3) opacities of the dioptric media, and (4) change in the choroid, retina and optic nerve. Thus myopes have an inaccurate impression of size, distance, color, and detail, and err in their judgments of matters concerning which their only or chief information comes through their organs of vision. So much is left to the imagination in the make-up of a landscape, a picture, a human face, that they suffer a rude awakening when made to realize by glasses the real appearance of things. The hypermetrope sees the detail, but misses the harmonious whole. The astigmatic individual sees the square an oblong, the circle an oval, and fails to perceive with equal clearness simultaneously all parts of an object (Compare Page 388.).

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[4] For more extended information on this subject the reader is referred to the recent papers of Drs. Wm. Campbell Posey and Howard F. Hansell.

The patient with a paralyzed ocular muscle receives two impressions of every object, neither of them correct, because the mental conception must be formed without the association of the binocular co-ordination and physiologic double retina image. The mental impression of opacities of the cornea is that of cloudiness; of opacities of the lens, that of a fog; of opacities of the vitreous, that of smoke. Minute scattered vitreous opacities are extremely deceptive, and lead the patient to believe he sees flies, spiders, webs, that elude his grasp as he reaches to brush them aside.

Irritation of the rods and cones of the retina and the axis cylinders in the nerve and retina by the pressure of blood or inflammatory material accentuates the normal activity of the tissues and the field of vision is occupied by brilliant meteoric displays of flashes and circles of light, bright or colored rings or waves of light rapidly passing from one side to the other, or emanating from the centre and spreading toward the periphery, startling and real while they last, and their disappearance is followed by a feeling of intense relief.

The author has notes of a number of cases in which patients had, without any evidence of nervous disease and exhibiting only faint opacities of the vitreous or slight choroidal lesions, distinct visions of rats or mice running across the room. In every instance these phenomena were visible towards one or other temporal side of the patient and at a distance of from two to three metres. As they all wore glasses the hallucination was attributed to images projected into the over-sensitive eye from the inner surface of one lens.

The scintillating scotoma, or "fortification spectrum," of *migraine* is sometimes seen without the occurrence of headache, vertigo or nausea. Occasionally it appears as a visual aura of epilepsy. In the first instance it may mislead the surgeon into the fear of an approaching optic atrophy, but an examination of the visual field and the use of the ophthalmoscope will clear the diagnosis.

Hemeralopia (sometimes called nyctalopia), or day blindness, constitutes a peculiar hyperæsthesia of the retina, in which vision is decidedly worse when the patient is exposed to bright daylight, but improves with a dim illumination. It is sometimes associated with true photophobia, phosphenes and with various other visual hallucinations. It is seen, now and then, after prolonged exposure to intense reflections from large areas of water, snow, ice, etc., and is not uncommon in albinism and in those suffering from coloboma of the iris and choroid.

Nyctalopia, on the other hand, occurs in a sensitive retina. The person so affected sees fairly well in ordinary daylight, but the moment the intensity of the illumination is reduced vision is much worse. This condition is occasionally functional, but is often due to organic disease of the choroid and retina, as seen in *retinitis pigmentosa,* syphilitic chorio-retinitis and in some degenerations of the retina not due to lues.

Erythropsia, or red vision, is one of the most frequent forms of chromatopsia. Colored vision is generally due to the absorption of pigments that either tinge the dioptric media or produce some unknown effect upon the percipient elements of the retina. Probably this explains the yellow vision (xanthopsia) of jaundice, as well as that following poisoning from santonin, amyl nitrite, cannabis indica, picric acid, etc. The complaint of aphakic patients that objects seen through the correcting lenses are blue, or deeply edged with that color, is not explained by the possible prismatic effect of the glasses. Probably the eye, accustomed to perceive objects through the yellowish medium of the lens, perceives them as blue (the complementary color of yellow) when the diseased crystalline is removed. Red vision follows exposure to extremely bright white or violet light, for instance, to the rays of the sun or to the light from the electric furnace.

AMBLYOPIA.

Amblyopia is a term indicating poor vision without apparent cause, particularly when the condition is not explained by an ophthalmoscopic examination.

Toxic Amblyopia.—The commonest forms of defective vision produced by poisoning are those from the habitual abuse of grain (ethyl) alcohol, tobacco in any of its forms, or from the use of both together. The disease is usually found in men over forty years of age who suffer from intestinal catarrh and who have an idiosyncrasy against stimulants. The patient complains of foggy or misty vision and generally applies to the ophthalmic surgeon for glasses to assist his failing sight. Central vision often falls to $\frac{2}{10}$ of normal, and the reading of ordinary print is impossible with any lens. Such a patient has a faulty digestion, an "old pipe" odor to his breath, anorexia and morning nausea, insomnia and dull headache. The ocular condition is mainly due to a central scotoma for red and green, the perimetric examination showing an oval defect about the fixation-point. The *prognosis* in this form of toxic amblyopia is generally very favorable, particularly if the patient can be persuaded to abandon his toxic habits.

Rarer forms of amblyopia from poisons are those produced by drinking *methyl* or *wood alcohol* ("Columbian Spirits," "Cologne Spirits," adulterated "Jamaica Ginger," lemon extract, bay rum, etc.), by the ingestion of quinine and by the absorption of lead salts. In all these cases complete blindness from optic atrophy may occur.

Reflex Amblyopia is a form of blindness due to irritation (generally peripheral) of the nerves in more or less close relation to the optic centres. Intestinal worms, especially in children, diseases of the teeth in adults and, in rare instances, pathologic changes in the reproductive organs have been held responsible for visual defects that have disappeared with the removal of the cause.

Amblyopia ex anopsia is a term of rather ill-defined meaning. In many cases of strabismus due to congenital conditions, or associated with them, the squinting eye, although amblyopic, presents no organic defect. However, after exercise of the weaker eye (following a removal of the barrier to binocular sight) not only central but peripheral vision of the eye may very greatly improve. The same statement is occasionally true of long-standing early cataract. It is not improper, then, to believe that the defective sight of such eyes was due to non-use of the organ—just as temporarily paretic muscles may undergo a functional atrophy and yet regain their original size on the return of normal innervation. It is not yet decided how one may unerringly differentiate between eyes capable of visual improvement and those that continue functionally incompetent during life in spite of all treatment.

Amblyopia from Toxins. Uræmia.—Loss of vision from drug poisoning has its counterpart in certain toxic conditions of the blood, particularly uræmia. The blindness, as in quinine and methyl alcohol amaurosis, comes on suddenly and the pupils are often dilated, although they generally react to light. There are no ophthalmoscopic changes due to the toxæmia proper, and sight returns when the state of the blood improves. Convulsions and other evidence of uræmia usually accompany the visual defect.

CHAPTER V.

THE EXTRA-OCULAR MUSCLES

By ALEXANDER DUANE, M.D.

Section I.

PHYSIOLOGY OF THE EYE-MUSCLES AND EYE-MOVEMENTS. TESTS. NOMENCLATURE AND CLASSIFICATION OF THE OCULAR DEVIATIONS.

Fixation and its Derangements.—**Monocular Fixation.**—When we direct one of our eyes towards an object so as to see it as distinctly as possible we are said to *fixate* or *fix* that object.

The fovea centralis is the most sensitive portion of the retina. Hence, in performing fixation we normally direct the eye so that the image of the object looked at falls upon the fovea. This is called *central fixation.* It may happen, however, that owing to disease of the retina or to other reasons vision with the fovea is less acute than with some outlying portion of the retina, and in that case the eye, in monocular fixation at least, will be directed so that the image shall fall upon this outlying portion (*eccentric fixation*).

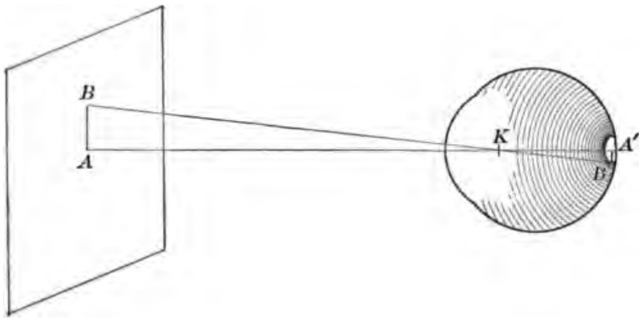
Monocular Projection.—When we are looking with one eye at an object and that object forms its image on the retina, we not only see the object, but get an idea that it occupies a certain definite position in space. This process by which we infer the position of an object in space from the visual impression that the object produces we call *projection.*

An object towards which the eye is pointing forms its image on the fovea. Conversely, an object which thus forms its image on the fovea we judge to be situated in that part of space towards which we think the eye is pointing. If our judgments are correct as to the direction of our eye, we project—*i.e.*, locate—objects correctly; but if, as in paralysis, our notions of the positions of the eye are erroneous, our projection will be faulty and we will see objects in the wrong place.

Not only do we get an idea of the location of an object, A (Fig. 1), towards which the eye is pointing, but also a notion, more or less precise, of the location of all objects surrounding A. Thus, an object, B, that forms its image below the fovea, we regard as situated in space correspondingly far above A, for, objects that are actually above the point we are looking at do regularly, in our experience, form their image below the fovea. So also the object, C (Fig. 2), which forms its image to the left of the fovea, we judge to be situated correspondingly far to to the right of A.

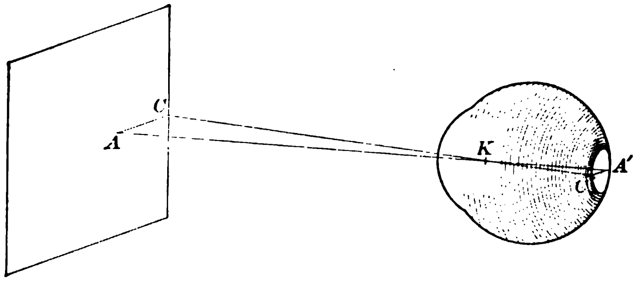
We thus arrive at the *law of projection*, which may be stated thus: ***An object which forms its image on any point in the retina is projected to*—i.e., *mentally located in—the point in space directly opposite.***

Fig. 1.



Binocular Fixation.—In the vast majority of cases, if one eye fixates an object with its fovea the other will do so too (binocular fixation). In fact, the whole extremely complicated mechanism of muscles and nerves with which the two eyes are supplied is designed primarily to accomplish this single end—*i.e.*, to so move the eyes that they shall both be directed accurately to the object we wish to see and each eye shall receive the image of that object precisely upon the fovea. The movements of the two eyes are hence almost invariably so co-ordinated as to secure binocular fixation under all conditions.

Fig. 2.



Derangements of Binocular Fixation; Deviations of the Eye.—Normally, as we have seen, a person looking with both eyes open fixates the same object with both,—*i.e.*, both eyes are " straight."

Not only is this so, but in the majority of cases both eyes will still fixate the same object,—*i.e.*, both will be "straight" even when one eye is covered. This condition is called *orthophoria*, and must be regarded as the ideal or natural condition.

In other instances both eyes will look straight at the same object when both are uncovered, but either eye as soon as it is covered will deviate—turning out, in, up, or down. This condition is called *heterophoria*, or, less properly, *insufficiency.*

In a third class of cases, comparatively few in number, only one eye is straight at a time, the other deviating even when both eyes are open. This condition is called *squint, strabismus*, or *heterotropia.*

Squint is further called—

I. (*a*) *Alternating* when sometimes one eye deviates, sometimes the other.
  (*b*) *Monocular* (right or left) when the deviation always affects the same eye.

Again, the deviation, whether squint or heterophoria, is called—

II. (*a*) *Intermittent* when under the same conditions it is sometimes present, sometimes not.
  (*b*) *Constant* when it is present all the time.
III. (*a*) *Periodic* when it is present when the eyes are directed at a near object and absent in distant vision, or *vice versa.*
  (*b*) *Continuous* when it is present both in distant and near vision.
IV. (*a*) *Comitant* or *concomitant* when the amount of deviation remains the same, and
  (*b*) *Non-comitant* when the deviation changes notably, as the eyes are carried in different directions.

A non-comitant deviation is usually of paralytic origin (paralytic squint or heterophoria). A comitant deviation is not usually due to paralysis, at least paralysis of the individual muscles, although it may develop out of such a paralysis.

Deviations are also named according to the *direction of the deviating eye.* Thus—

HETEROPHORIA.

Both eyes fix when both are uncovered, but behind screen—

Either eye turns in	Esophoria.
Either eye turns out	Exophoria.
R. eye turns up and L. down *	R. Hyperphoria.
L. eye turns up and R. down *	L. Hyperphoria.

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* In some anomalous cases (see Section VI.), instead of one eye going up and the other down when screened, each eye goes up or each down.

SQUINT.

When both eyes are uncovered one fixes and the other turns—

In ......Esotropia (convergent squint, strabismus convergens), R. or L.

Out .....Exotropia (divergent squint, strabismus divergens), R. or L.

Up......Hypertropia (upward squint, strabismus sursumvergens), R. or L.

Down....Hypotropia (downward squint, strabismus deorsumvergens), R. or L.

Behind screen—

Either eye deviates in...........Esotropia (R. or L.).

Either eye deviates out..........Exotropia (R. or L.).

R. eye turns up and L. eye down [*]...R. Hypertropia or L. Hypotropia.

L. eye turns up and R. eye down [*]...L. Hypertropia or R. Hypotropia.

Esophoria, exophoria, esotropia, and exotropia are also known as *lateral* deviations (heterophoria, squint); hyperphoria, hypertropia, and hypotropia as *vertical* deviations.

Mixed conditions,—e.g., combinations of a vertical with a lateral deviation,—are quite frequent. Especially common are the conditions prevailing in a developing squint (see Section IV.), in which heterophoria or orthophoria alternate with a true strabismus, or a squint for near is associated with heterophoria for distance, etc. Lastly, a patient, especially in paralytic cases, may show an esotropia if he looks in one direction and an exotropia if he looks in another; or, he may have an esotropia for distance and an exotropia for near, as in a combined divergence- and convergence-paralysis (page 257); or he may have an esotropia if he fixes with one eye and exotropia if he fixes with the other. In the case last mentioned, one eye will deviate *out* when covered, the other eye will deviate *in* (Graefe). (See Page 251, note.)

Binocular Single Vision: Fusion.—As we have seen, in all but a small minority of cases binocular fixation is present whenever we look with both eyes. The image of the object looked at then is formed on each fovea. Yet, though there are the two images, we see, not two objects, but one (binocular single vision). For the mind by a peculiar process which we denominate *fusion* combines the two retinal images into a single visual impression. This binocular visual image differs somewhat in character from the monocular visual image gained when we look with either eye by itself, being rather more distinct and also more solid-looking. In fact, vision with both eyes differs from vision with one eye in that with the former we get clear, and with the latter but imperfect, ideas of distance, depth, and relief. In other words, when we look with both eyes we have solid or *stereoscopic vision.*

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[*] In some anomalous cases (see Section VI.), instead of one eye going up and the other down when screened, each eye goes up or each down.

The mental faculty by which fusion is effected is called the *fusion faculty.* It acts, presumably, by means of a cerebral nerve-centre (*fusion centre*), the site of which, however, is altogether undetermined. Absence or non-development of the fusion faculty is thought to be an important factor—according to some, the prime factor—in the production of squint.

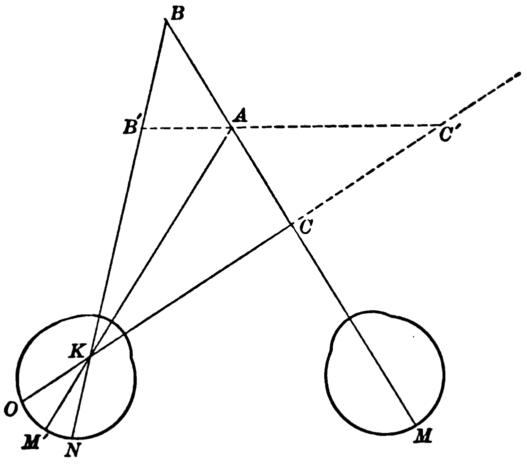
Binocular Projection.—The visual impression produced by this fusion of the two retinal images is by the mind projected, or referred to a portion of space outside of the body, being thus regarded as a something apart from ourselves and occupying a certain place in the exterior world. The point to which we thus refer it is the point of intersection of our two visual lines. Unless the eyes abnormally deviate, this point corresponds to the actual situation of the object itself as determined by our other senses. Thus, when we look at an object one foot off, the eyes turn until their visual lines are both directed at the object, and hence intersect at the point where it is situated. Then each foveal image is projected out to this point of intersection,—*i.e.*, to the place that the object itself actually occupies.

Corresponding Points.—Not only does the mind fuse the two retinal images of any object looked at directly, so that that object appears single, but it fuses the images of a good many other objects as well. Thus, if we are fixing an object A with both eyes, A, which forms an image on each fovea, will appear single and will look to be straight in front of the eyes. Another object, B, situated to the right of A, will form its retinal image a certain distance, say 10° to the left of the fovea in the right eye. If now it is so situated as to form its retinal image 10° to the left of the fovea in the left eye too, it will appear single, the two outlying retinal images being fused in this case just as the two foveal images were. In general, any object whose retinal images are formed equally far and in the same direction from the fovea in each eye—are formed, as we say, on the *corresponding points* of the two retinas—will appear single.

Diplopia.—As we have just seen, when the images of the same object fall upon corresponding points, the object appears single. It is otherwise when the images fall upon points not corresponding,—*e.g.*, on the fovea in one eye and on any outlying portion of the retina in the other. Then, in general, we have double vision (diplopia), the mind receiving two visual impressions of the same object. In this case each eye projects the image differently,—*i.e.*, locates it in a different point in space.

Physiological Diplopia.—In many cases this diplopia is physiological. Thus, in Fig. 3 the object A, at which both eyes are looking and which, therefore, forms its retinal images on the two foveæ M and M′ respectively, appears single, being projected by both eyes to its true place, A; but B, an object more remote than A and situated in the prolongation of the line MA, will appear double, for while it forms its image in the right eye on the fovea, M, it will form its image in the left eye on a point, N, to the right of the fovea. To the right eye it will appear directly in line with A (behind it); to the left eye it will, according to the law of projection, appear to be to the left of A, and the more so the further N is to the right of M′. We shall have thus a condition of ***homonymous*** diplopia,—*i.e.*, a condition in which the image seen by the left eye is to the left of that seen by the right eye.

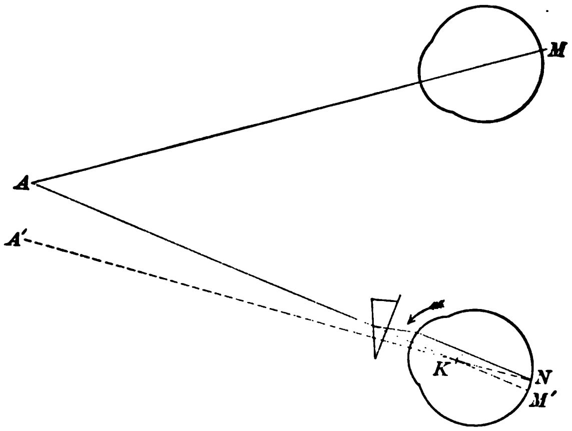
FIG. 3.



The object, C, lying nearer to the eyes than A and in the line, AM, will, to the right eye, seem to be in line with A (just in front of it) and to the left eye will seem to lie to the right of A, since C forms its image in the left eye at O to the left of the fovea. Such diplopia, in which the image seen by the left eye appears to the right of that seen by the right eye, is said to be *heteronymous* or *crossed.* This physiological diplopia, crossed for objects nearer than the object looked at and homonymous for more distant objects, although readily demonstrable, rarely, if ever, appears spontaneously and hardly ever occasions any confusion.

Artificial Diplopia produced by Prisms.—Diplopia may also be produced by prisms. Thus, suppose both eyes are looking at an object, A (Fig. 4) so that the image of that object falls on the fovea in each eye, and then a prism with the *base directed inwards* (towards the nose) is placed before the left eye. The prism will deflect the rays of light towards its base, so that the retinal image in the left eye will no longer fall on the fovea, M′, but to the right of it, N. The eye, according to the law of projection, will regard this image as situated to the left of A,—*i.e.*, the right eye fixing A with its fovea will see A in its true place, while the left eye will see a similar image, but dislocated to the left (at A′). There will then be a condition of homonymous diplopia.

Fig. 4.

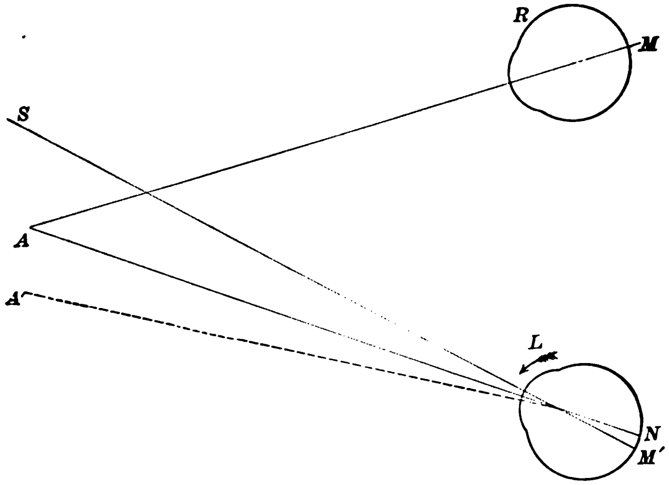


So also, a prism placed *base out* before one eye will produce crossed diplopia. A prism placed *base up* or *base down* will make the corresponding image appear below or above the other,—*i.e.*, will cause a vertical diplopia.

Pathological Diplopia.—Diplopia is also caused by a deviation of the eyes. That is, if the right eye (R. Fig. 5) is fixing the object A, but the left eye, instead of fixing it, is *turned too far inwards*, the retinal image of A in this eye will fall not on the fovea M′, but to the inner or right side of it (N). Now, the mind really does not know how the left eye is pointing; in fact, it seems to regard both eyes as looking in the same direction; hence, an object whose image is on the fovea of the left eye will be projected out to A, the direction in which the mind conceives this eye to be pointing (page 178), and an object whose retinal image is at N, to the right of the fovea, will be regarded as located correspondingly far to the left of A. Such a patient, therefore, sees two images of A—one seen by the right eye, situated in its true place straight ahead; the other seen by the left eye, situated to the left (A′)—*i.e.*, there is here an homonymous diplopia. If the left eye had been *turned out* there would have been a crossed diplopia; if *up* or *down*, a vertical diplopia.

Overcoming Diplopia.—Diplopia produced by prisms, or diplopia produced by pathological deviation, may be overcome if the eye is turned so that the image in either case falls upon the fovea. Thus, the homonymous diplopia shown in Fig. 4 and in Fig. 5 will be abolished if the left eye turns out as shown by the arrow, for, turning the eye out will turn the fovea M′ in, and when M′ has gone in far enough the retinal image of A will fall upon it; then the retinal image will fall on the fovea in each eye, and there will be single vision (See Page 181).

FIG. 5.



The ability to overcome diplopia depends partly upon the readiness with which the various movements required for the purpose can be executed, partly upon the development of the fusion faculty (See Page 182). A man, however well developed and well regulated his eye muscles and the nerve-centres that co-ordinate them may be, will not unite double images well if his fusion faculty is imperfect. Ill development or temporary abeyance of the fusion faculty probably accounts in part for the fact that in some cases of neurasthenia, hysteria, and organic brain disease, diplopia is so easily produced and when produced is so difficult to overcome. In such cases the mere act of placing a red glass or a 1° prism before one eye suffices to evoke an insuperable diplopia. Again, there are some persons who never by any adjustment of the eyes or with any prism get fusion of the double images,—in whom, for instance, a prism of 6°, base out, produces a crossed, and a prism of 5°, base out, a homonymous diplopia, but no prism produces single vision. This condition of so-called *antipathy to single vision* occurs in squint, particularly after operation, and is probably attributable to non-development of the fusion faculty.

Varieties of Diplopia.—The foregoing facts, together with the nomenclature of the different varieties of diplopia are succinctly shown in the following table:

TABLE OF DIPLOPIA.*

NAME OF DIPLOPIA		CHARACTER	CAUSED BY		CORRECTED BY	
		Image of R. eye as compared with that of L. is	(1) A natural deviation of	(2) Artificially by a prism placed base.	(1) Turning.	**(2) Prism placed with base.**
LATERAL.	Homonymous.	On the right.	Either eye *inward* (esophoria, esotropia).	*In*, before either eye.	Both eyes *outward*. (Divergence.)	***Out* before either eye.**
	Heteronymous. (Crossed.)	On the left.	Either eye *outward* (exophoria, exotropia).	*Out*, before either eye.	Both eyes *inward*. (Convergence.)	***In* before either eye.**
VERTICAL.	Right.	Below.	R. eye *up* or L. eye *down* (R. hyperphoria, R. hypertropia, L. hypotropia.	*Up* before R. eye, *down* before L. eye.	R. eye *down* and L. eye *up* (L. supravergence).	***Down* before R. eye or up before L. eye.**
	Left.	Above.	R. eye *down* or L. eye *up* (L. hyperphoria, L. hypertropia, R hypotropia).	*Down* before R. eye, *up* before L. eye.	R. eye *up* and L. eye *down* (R. supravergence).	***Up* before R. eye or *down* before L. eye.**

In conditions producing vertical diplopia, especially when the eyes are directed down, one image (usually the one that should be higher) often seems to be on a level with the other, but back of it. This is due partly to the character of the background upon which the images are projected, partly to other factors (M. Sachs, Frölich).

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* Modified from the author's article in De Schweinitz and Randall's "American Text-Book of the Eye, Ear, Nose, and Throat."

Character of the Images in Diplopia.—In diplopia the image seen by the fixing eye is usually sharp and clear because that eye sees it with the fovea, the portion of the retina having the most distinct vision. This image is hence called the *real* image. The other, or *false*, image, projected by the non-fixing eye, looks shadowy and indistinct, because it is seen by an extrafoveal portion of the retina.

Binocular Fixation and Binocular Single Vision compared.—*Derangements of binocular fixation* cause the various deviations of the eye (squint or heterophoria). Such a derangement is proved to exist if the deviation can be made out objectively,—*i.e.*, if there is a deflection of the eyes which the observer can discover on inspection of the patient.

On the other hand, *disturbance of binocular single vision is* proved to exist if the patient himself has the subjective sensation of diplopia.

Upon these two facts depend all of our tests, which are hence classed as *objective* when they show disturbance in binocular fixation which the observer can detect, and *subjective* when they depend directly or indirectly upon the demonstration of diplopia.

The two conditions—disturbance of binocular fixation and disturbance of binocular single vision—usually occur together and are equally marked. In this case the objective and subjective tests can both be employed and will, moreover, agree with each other. In two classes of cases, however, this is not so:

I. In most cases of old squint *binocular vision is absent*—that is, when both eyes are open the image formed on the retina of the squinting eye is mentally suppressed so that the patient sees with the other eye only. When the squinting eye alone is used for seeing, this suppression does not occur; hence if a red glass is placed before the squinting eye and the patient looks at a light with his right eye shut he will see a red light, while if he either has both eyes open or has the left eye shut he will see simply a white light. In these cases, therefore, the patient cannot be made to recognize diplopia; hence, all the subjective tests which in one way or another depend upon the recognition of diplopia, fail, and we have to depend entirely on the objective tests (those for binocular fixation).

II. In many cases of squint, and particularly of cured squint, both *subjective and objective tests are applicable, but are discrepant* because the patient projects or locates objects in space erroneously with his squinting eye. Thus, we may have a condition in which the eye is obviously turned in, and yet there is either a crossed diplopia, or, if there is a homonymous diplopia it does not correspond in amount to the degree of deviation (incongruity of the double images).

Muscles of the Eye.—The muscles moving the eyeball and eyelids are the internal, external, superior, and inferior recti, the superior and inferior obliques, and the levator palpebræ.

The *four recti* and the *levator* arise from the lesser and greater wing of the sphenoid and from the dense fibrous membrane filling in the sphenoidal fissure. Their combined origins form a tendinous ring which surrounds the optic foramen and that portion of the sphenoidal fissure giving passage to the oculomotor and abducens nerves. These five muscles pass forward, diverging as they go, so as to form a hollow cone (muscle cone), which includes the second, third, and sixth nerves, and the ciliary ganglion.

The superior rectus lies underneath the levator, to which it is connected quite firmly by bands of fascia. The other muscles at first run quite close to the walls of the orbit, but somewhat in front of the middle of their course the four recti turn in toward the eyeball, and piercing the orbital fat and Tenon's capsule, are inserted into the sclera around the cornea. The internal rectus is inserted 5.5 millimetres, the inferior 6.5 millimetres, the external 7 millimetres, and the superior 7.5 to 8 millimetres from the corneal margin. The levator passes on to be inserted into the tarsus of the upper lid.

The *superior oblique* arises from the body of the sphenoid, runs forward, keeping close to the upper wall of the orbit, and at the upper inner margin of the orbit passes through a tendinous ring (the trochlea), from which it is reflected obliquely backward and outward so as to pass beneath the tendon of the superior rectus. It pierces Tenon's capsule, and is inserted into the upper outer portion of the eye back of the equator.

The *inferior oblique* arises from the inner lower wall of the orbit just back of the nasal duct, passes obliquely up and out beneath the inferior rectus, and is inserted into the lower outer part of the eyeball behind the equator.

Mention has been made of *Tenon's capsule.* This is a spherical capsule of connective tissue surrounding the whole eye except the cornea and a little rim of sclera around it in front, and the optic nerve and a little annular area of sclera about it behind. It is thus like a broad convex ring surrounding the eye like a sling. The anterior margin of this ring is fused with the conjunctiva, and both conjunctiva and capsule are attached firmly to the sclera round the cornea. The posterior margin of the capsular ring is also attached to the sclera, so that the space between the eyeball and Tenon's capsule is closed in, both anteriorly and posteriorly. This space, called *Tenon's space,* is traversed by connective tissue bands joining the capsule to the eyeball. Tenon's space is held to be the analogue of a joint cavity, although it is filled neither with synovia nor with lymph. This analogy must not be pushed too far, for while the eye does, in fact, turn in the orbital tissue as in a socket, it is probable that as Tenon's capsule must, to a certain extent, move with the eye to which it is attached, the real socket of the eyeball lies in the orbital fat outside of the capsule (Motais, cited by Maddox).

In considering the action of the muscles, we have to take into account not only the primary insertion (to the eyeball), but also the secondary insertion (to the orbital walls and eyelids).

The *primary insertion* of each muscle is by a broad, flat tendon, implanted into the sclera beneath the conjunctiva and Tenon's capsule. This insertion is reinforced by a fibrous band on each side connected with the sclera and extending about one to one and one-half millimetres in front of the insertion proper. These *adminicula*, as they are called, like the insertion itself, lie beneath Tenon's capsule,—*i.e.*, within the capsular cavity. Other bands are given off from the tendon just before it enters the capsule which attach the tendon to the outer surface of the latter.

The *secondary insertions* of the tendons are:

I. Check ligaments or firm bands of fascia extending from the anterior portion of the muscles to the orbital walls.

II. The palpebral bands extending to the septum orbitale and the lids.

The *check ligament* of the internal rectus unites with that of the inferior rectus and is inserted into the lachrymal bone behind the lachrymal sac.

The external rectus has two check ligaments,—one below, the other above,—both attached to the outer wall of the orbit near its margin.

The superior rectus and levator palpebræ have two check ligaments. The outer, broad and firm, fuses with the upper check ligament of the externus and forms a firm septum supporting the lachrymal gland, and is attached to the upper wall of the orbit near its margin. The inner check ligament is attached to the upper, inner part of the orbit as far forward as the trochlea.

The superior oblique has two check ligaments—one, the posterior, running from the belly of the muscle to the trochlea; the other, the anterior, running from the trochlea backward upon the reflected tendon to which it is attached.

The inferior oblique has no proper check ligament. It is attached by very strong fibrous bands to the inferior rectus, the fascia here being so dense and strong as to act like a sling supporting the eyeball (hence, called by some the *suspensory ligament* of the eye).

The *action of a check ligament* is twofold: first, it restricts the action of the corresponding muscle so as to limit very considerably the rotation the latter would otherwise produce; second, it prevents the recti muscles from retracting the eyeball into the orbit. In fact, the eyeball, because it is held by the various check ligaments, and also because it is pulled in opposite directions by the obliques (which tend to draw it forward), and by the recti (which tend to draw it backward), is kept slung in one position, so that, instead of advancing or receding, it simply rotates in its socket.

The place of a check ligament for the inferior oblique is taken by the anterior ligament of the superior oblique which becomes tense in proportion as the inferior oblique contracts and the superior oblique relaxes.

The *palpebral bands* are the superior, running to the tarsus of the upper lid from the fascia connecting the superior rectus with the levator palpebræ, and the inferior, running to the tarsus of the lower lid from the fascia between the inferior rectus and the inferior oblique. When the superior rectus contracts, the superior palpebral band is pulled upon and the upper lid is elevated so that lid and eye rise together. So also when the inferior rectus contracts, the lower lid is pulled downwards by the inferior palpebral band.

Actions of the Ocular Muscles.—The actions of the eye muscles may be divided into main and subsidiary.

1. The **main actions** are as follows: The *external rectus* abducts the eye,—*i.e.*, moves it straight outwards or towards the temple.

The *internal rectus* adducts the eye,—*i.e.*, moves it straight in or towards the nose.

The *superior rectus* elevates the eye. As the muscle runs from within outward, its elevating action will be greatest with the eye abducted, because then it will be in line with the anteroposterior axis of the eye and will thus work to the greatest advantage. On the other hand, when the eye is adducted the muscle will be nearly at right angles to the anteroposterior axis of the eye, and will hence have little or no action as an elevator.

The *inferior oblique* acts to pull the back part of the eye down, and hence throws the front part up. Like the superior rectus, therefore, it is an elevator of the eye. As it runs quite obliquely backward and outward its elevating action will be greatest when the eye is adducted, for then the muscle will be nearly in line with the anteroposterior axis of the eye. On the other hand, when the eye is abducted its elevating action will be practically *nil*.

The *inferior rectus* depresses the eye, and, like the superior rectus to which it is nearly parallel, it will depress the eye most energetically when the latter is abducted, and little or not at all when it is adducted.

The *superior oblique* elevates the back part of the eye, and hence throws the front part down like the inferior rectus; therefore, it is a depressor, but, owing to the oblique way in which it runs, it depresses the eye the most when the latter is adducted, and little or not at all when it is abducted.

The *levator* lifts the upper lid.

2. The **subsidiary actions** of the eye are as follows:

I. *Lateral Action of Vertical Muscles.*—The superior and inferior recti, owing to the obliquity of their course, adduct the eye, and the more so when the eye is already adducted.

The two obliques, for a similar reason, abduct the eye, and the more so the more the eye is already abducted.

II. *Torsion Movements.*—The superior rectus and the superior oblique intort the eye,—*i.e.*, rotate the top of its vertical meridian inward, or towards the nose.

The inferior oblique and inferior rectus extort the eye,—*i.e.*, rotate its vertical meridian outward.

The intorting action of the superior rectus and the extorting action of the inferior rectus are most pronounced when the eye is adducted; the intorting and extorting action of the obliques is most pronounced when the eye is abducted.

III. *Action of Eye-Muscles on Lids.*—The superior rectus by virtue of its palpebral band elevates the upper lid, and the inferior rectus depresses the lower lid.

Monocular Movements.—From what has just been said it is evident that the eye is *adducted* mainly by the internal rectus, assisted towards the end of its course by the superior and inferior recti.

It is *abducted* by the external rectus, reinforced towards the end of its course by the two obliques.

It is *elevated* by the superior rectus and inferior oblique. The superior rectus acting alone would carry it up and a little in; the inferior oblique acting alone would carry it up and a little out; the two muscles acting together would carry it straight up. It is quite certain from the results, both of physiological experiment and the data derived from cases of paralysis, that when we look up both muscles do always act together. When we look *straight up*, both act quite strongly as elevators, although the superior rectus, having the less oblique course, is rather more efficient than the inferior oblique. When we look *up and out*, although both muscles still act, the superior rectus is the only one that lifts the eye, the superior oblique simply tending to abduct it still more and thus reinforce the action of the external rectus. *Per contra*, when we look *up and in*, the inferior oblique is the main agent in lifting the eye, the superior rectus simply acting to carry the eye inward and so reinforce the action of the internal rectus.

In all movements of elevation of the eye, whether straight up, up and in, or up and out, the upper lid is simultaneously elevated by the levator and the superior rectus.

The eye is *depressed* by the inferior rectus and superior oblique. The inferior rectus acting alone would carry it down and a little in; the superior oblique down and a little out. As a matter of fact, both muscles always act together and the eye is carried *straight down* through their combined action, the inferior rectus being rather the more effective of the two. When we look *down and out* the inferior rectus carries the eye down and the superior oblique carries it out, reinforcing the action of the externus. When we look *down and in* the superior oblique carries the eye down, the inferior rectus acting simply to carry the eye in and reinforce the action of the internal rectus.

In all movements of depression of the eye, whether straight down, or down and in, or down and out, the lower lid is simultaneously depressed by the inferior rectus.

Movements of the Lids.—The *upper lid is raised* by the levator, assisted by an unstriated muscle, the superior muscle of Müller, which is supplied by the sympathetic. Moreover, when the eye is elevated, so that the superior rectus is brought into play, this muscle also takes part in raising the lid.

The *lower lid is depressed*, partly by its own weight, partly by the contraction of the unstriated inferior muscle of Müller, which is supplied by the sympathetic. Moreover, when the eye is depressed, so that the inferior rectus is brought into action, this muscle also takes part in depressing the lid (See Chapter VII).

Field of Fixation.—By the action of the several muscles, either single or combined, the eye is able to make excursions in every direction—up, down, in, out, and obliquely. In this way, without moving his head, one can fix in succession all the objects occupying a large portion of the space about him. By thus moving the eye as far as possible in every direction, we delimit what is called the field of fixation. (See Section on Tests.)

Position of Equilibrium, or Primary Position.—When all the muscles that move the eye are at rest, or at least when no one of them is contracting more than any of its fellows, the eye assumes its position of equilibrium, or what is called the primary position. This position, in the great majority of cases, is such that the line of sight is perpendicular to the line connecting the centres of rotation of the two eyes, and lies in or slightly below the horizontal plane,—*i.e.*, the eye looks right ahead and on the level or a little below it.

Field of Action of the Muscles.—As we have just seen, the external rectus acts to carry the eye directly outward. When the eye is turned in, this muscle relaxes, and the only effect it produces is that due to the passive traction exerted by its tendon. Thus, the external rectus acts only in the outer half of the field of fixation. This outer half of the field of fixation we may then denominate the field of action of the external rectus. Similarly the field of action of the superior rectus will be the upper half of the field of fixation, and if we have regard only to the elevating action of the superior rectus, its field of action will be confined mainly to the upper outer quadrant of the field of fixation.

From a similar consideration it is evident that each one of the six muscles moving the eye has its special field of action, as shown in the following table:

MUSCLE.	SPECIAL FIELD OF ACTION.
External rectus.	Outer half of field of fixation.
Internal rectus.	Inner half of field of fixation.
Superior rectus (as an elevator).	Upper and outer quadrant of field of fixation.
Inferior oblique (as an elevator).	Upper and inner quadrant of field of fixation.
Inferior rectus (as a depressor).	Lower and outer quadrant of field of fixation.
Superior oblique (as a depressor).	Lower and inner quadrant of field of fixation.

Binocular Movements.—The two eyes when acting together cannot move independently of each other. The whole purpose of the neuromuscular apparatus of the eyes is to secure binocular fixation. Hence, movements which will interfere with binocular fixation are not ordinarily performed, and, more than that, cannot be performed except to a very limited degree. The movements actually possible to the two eyes working together are as follows:

I. **Parallel Movements.**—This class of movements is employed when we carry the eye from one distant object to another. In this case the two visual lines remain parallel with each other, both moving equally far and equally fast to the right, to the left, up, down, and obliquely.

An analysis of the statements made regarding the actions of the muscles for moving each eye individually (see Section on Monocular Movements) will show that the twelve muscles moving both eyes may be classed as follows:

A. LATERAL ROTATORS.

Producing lateral rotation of both eyes—*lateriversion.*[*]

(a) *Right rotators* (acting when the eyes are directed to the right).	(b) *Left rotators* (acting when the eyes are directed to the left).
Producing rotation to the right (*dextroversion*).	Producing rotation to the left (*levoversion*).
R. External rectus.	R. Internal rectus.
L. Internal rectus.	L. External rectus.

B. ELEVATORS.

Producing elevation of both eyes (*sursumversion*).

(a) *Right-handed elevators* (acting mainly when the eyes are directed to the right).	(b) *Left-handed elevators* (acting mainly when the eyes are directed to the left).
R. Superior rectus.	R. Inferior oblique.
L. Inferior oblique.	L. Superior rectus.

C. DEPRESSORS.

Producing depression of both eyes (*deorsumversion*).

(a) *Right-handed depressors* (acting mainly when the eyes are directed to the right).	(b) *Left-handed depressors* (acting mainly when the eyes are directed to the left).
R. Inferior rectus.	R. Superior oblique.
L. Superior oblique.	L. Inferior rectus.

It will be seen that the twelve muscles are divided into three groups, and each group comprises two pairs, the muscles of one pair acting when the eyes are directed towards the right, the muscles of the other pair acting when the eyes are directed to the left.

This makes six pairs of muscles—one muscle of each pair being in the right eye, the other in the left. The two muscles constituting a pair are called *associates.* Two associates acting together will move the right eye and the left eye respectively in the same direction and to the same extent. Thus, the right inferior rectus moves the right eye down, to the left, and rotates its vertical meridian to the right. Its associate, the left superior oblique, moves the left eye down, to the left, and rotates its vertical meridian to the right.

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[*] In lateriversion the movements of abduction made by one eye and of adduction made by the other are called *latero-abduction* and *latero-adduction,* respectively (Schapringer). They are to be distinguished from divergence-abduction and convergence-adduction (pages 195, 196).

The precision with which parallel movements are performed is remarkable. Under ordinary circumstances, parallelism is maintained up to the limits of excursion of the eyes in any given direction. If this is not the case—if one eye for any reason fails to keep pace with the other in advancing in any particular direction—diplopia ensues. The point where such diplopia begins indicates the limits of binocular single vision in that special direction of the gaze, and by determining these limits for all directions of the gaze—right, left, up, down, and obliquely—we can map out what is called the *field of binocular vision.*

As we shall see later, any marked restriction of the field of binocular single vision in some one direction of the gaze, indicates an insufficiency or paralysis of one of the ocular muscles.

There appears to be a distinct *nerve-centre* or tract governing each one of the different parallel movements of the eyes,—*i.e.*, there is one centre governing the movements to the right, one governing the movements to the left, one governing upward movements, etc. The centres for the initiation of such movements (*centres of voluntary movement*) are apparently in the frontal lobe (Russell, Mott and Schäfer, Sherrington, Topolanski). In addition to these there are posterior cortical centres which are undoubtedly situated in the angular gyrus (Bernheimer). These are *reflex centres,*—*i.e.*, designed for movements set up in a reflex way by visual impressions. Stimulation of either the anterior or the posterior cortical centres on the right side produces a movement of both eyes to the left (or up and to the left or down and to the left), and, if very strong, also produces *inhibition of the movement to the right.* If the stimulus starts from the angular gyrus, it crosses over just beneath the aqueduct to reach the *subcortical centres* which are found in and about the nerve nuclei of the opposite side. The thalami and corpora quadrigemina take no part in the production of parallel movements (Topolanski).

II. **Movements of Convergence.**—When we pass from a distant object to one nearer we have to converge the eyes. We do this by means of a simultaneous and equal contraction of the two internal recti, producing an equal movement inward (*convergence-adduction*) of each eye. This movement of convergence may be combined with a lateral or vertical parallel movement, as when we look at a near object that is situated to the right or left, or is situated above or below the horizontal plane.

The *centre for the initiation of convergence-movements* (voluntary movements of convergence) is probably in the frontal lobe (according

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with both eyes open, and then alternately cover and uncover one eye,—say the right,—leaving the other all the time uncovered (*method of binocular uncovering*). By thus covering the right eye we compel the left eye to fix, if it is not already doing so, and we allow the right eye to deviate if it has any tendency to do so if it is not already deviating.

In this case three conditions may obtain:

(*a*) There may be *heterophoria*. In this event the left eye which is fixing already will continue fixing when the right eye is covered, and the latter which was fixing before being covered will not deviate. Then, when the cover is taken off again, the right eye will swing back into place and the left will still remain fixing,—*i.e.*, both on applying and on removing the screen but one eye will move, and that the right.

(*b*) There may be a *squint of the right eye*. In this case the left eye which was fixing before the right was covered will continue fixing afterwards, and the right eye which was deviating before it was covered will remain deviating afterwards. Then, on uncovering, the left eye will still remain fixing and the right will still remain deviating,—*i.e.*, both on applying and on removing the screen neither eye moves, provided the screen is put over the squinting eye.

(*c*) There may be a *squint of the left eye*. Then the left eye, which was deviating before the right eye was covered, will now have to move into place in order to fix. As it moves into place, the right eye, which is behind the screen, will move out of place. Then, when the screen is removed, the right eye which has thus become deviated, will move back into the fixing position, since it is the right eye which ordinarily fixes when both eyes are open, and the left eye will move out of the fixing position and back to its ordinary position of deviation,—*i.e.*, both on applying and on removing the screen both eyes will move, provided in the case of a squint the screen is put before the fixing eye.

If repeated applications of the test show that sometimes the right eye squints, sometimes the left, then we are evidently dealing with an *alternating strabismus*.

III. **Tests of Binocular Vision.**—To determine whether the patient has binocular vision or not, and, in case he has binocular vision, whether there is diplopia or a tendency to diplopia, we employ a large number of tests.

As has already been said, the tests of binocular vision *do not agree with those for binocular fixation*,—*e.g.*, with the screen test,—if there is any incongruity of the double images (See Page 187).

Again, if there is suppression of the image in one eye, such as regularly takes place in squint, *all the tests for binocular vision will*

a pencil or a light, held straight in front of him. Inspectio
show, approximately at least, whether his fixation is central
or whether, as in cases of a high grade amblyopia, he can
but allows his eye to wander aimlessly, getting mere gli
object shown him.

II. **Tests of Binocular Fixation.**—Of the several tests
fixation, by far the best and most reliable is the *scree*
alone will be described here. In performing this we
look at an object fifteen feet or more from the eyes.

The patient is directed to look sharply at the ob
light), and then the card is placed before the rig
quickly to and fro from one eye to the other.

If there is *orthophoria*, neither eye will deviate
each consequently will remain steady when the scr
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when covered will deviate perceptibly, and when
back into the fixing position. Thus, if there is
convergent squint each eye in turn will deviate
when covered, and will swing out again when

Whether the two eyes deviate to the same a
screen will depend upon which has the strong
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left, and in the case of spasm of the left eye w
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than that of the left. (Compare remarks u
Section II., and Symptoms of Spasm, Sec
occurs in certain cases of squint. (See
Heterophoria and Squint, Section IV.)

The *amount of the deviation* can be
of the prism that abolishes it. Thus, if
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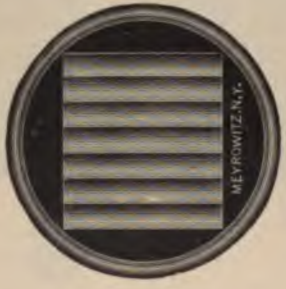
come the diplopia. The latter method, however, usually gives an insufficient measure of the amount of the diplopia. The real diplopia, for example, may be 20° and yet be apparently nullified by a prism of 18°, because such a prism brings the images so close together that the patient then unites them himself without further aid.

In testing for diplopia in this way we ask the patient not only how the double images are placed with regard to each other and how far apart they are, but also whether both are upright or not. If *one of the images is tilted*, either to the right or to the left, it indicates that the corresponding eye is tilted and has its vertical meridian rotated in the opposite direction. Thus, if the candle seen by the right eye tilts to the right, it means that the right eye itself is tilted to the left or has a pathological intorsion.

3. *Maddox Rod.*—The Maddox rod consists of one or more little cylinders of glass set in a frame (Fig. 6). If the instrument is held as shown in the figure and placed before the right eye, the patient, looking at the light, will see with this eye the light transformed into a luminous vertical streak. If he has neither esophoria nor exophoria this streak will appear to run through the flame which in its unaltered form he sees with the left eye. If he has esophoria it will appear to the right of the flame; if he has exophoria, to the left of it.

Fig. 6.



If now the rod is turned at right angles to the position shown in the cut and again placed before the right eye, the patient, if he has no hyperphoria, will see a horizontal line bisecting the light. If, however, he has right hyperphoria the horizontal line will appear below, and if he has left hyperphoria it will appear above the light.

The *amount of deviation* may be measured by the linear distance of the streak from the light (measured on a tangent scale) or by the amount of prism that will be required in order to make the streak coincide with the light.

The Maddox rod can also be used to indicate *whether there is any torsion* of the eye,—*i.e.*, any tilting of its vertical meridian to the right or left. If there is, the streak of light will appear tilted when the rod is set directly vertical, and the number of degrees through which the rod has to be rotated in order to make the line of light appear vertical, will indicate the amount of the torsion. Thus, if the vertical meridian of the right eye is rotated 10° towards the temple, we shall have to turn the Maddox rod out 10° in order to make the line of light appear vertical.

4. *Phorometer.*—The phorometer is an instrument for producing artificial diplopia by means of prisms, and from the relation of the double images deducing whether there is any tendency to deviation or not.

Stevens's phorometer consists essentially of two 6° prisms, geared so that they can be rotated simultaneously but in opposite directions. The prisms are first set so that their bases both look inward; the patient then looking through the prisms will see two images of the test object, that of the right eye being on the right side. If he has no hyperphoria the images will be on the same level. If he has right hyperphoria the right image will be lower; if he has left hyperphoria it will be higher.

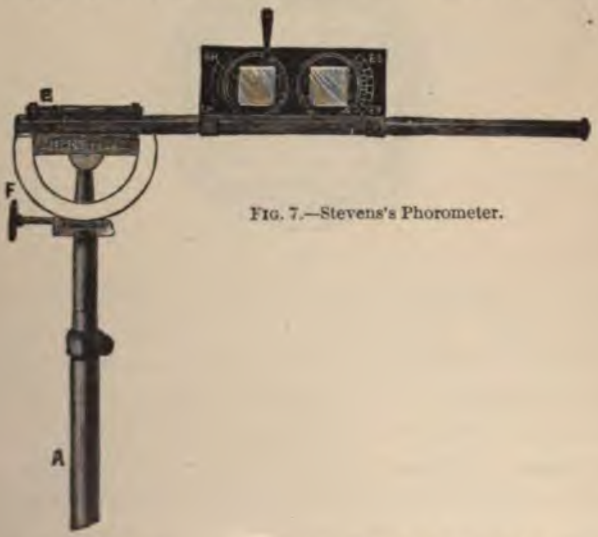


FIG. 7.—Stevens's Phorometer.

In case the images are not on a level the prisms are rotated until they are; then the amount of hyperphoria can be read off from the index.

To measure for lateral deviations the prisms are rotated until the right-hand one points down, the left up. Then, if there is no esophoria or exophoria, the images will appear directly over each other. If, however, there is esophoria the upper image will appear on the left; if there is exophoria it will appear on the right. In either of the latter events the prisms are rotated until the images do stand over each other, when the amount of the esophoria or exophoria, as the case may be, can be read off from the index.

5. *Bar-reading.*—If the patient is directed to read and holds a pencil some three inches in front of the page, between it and his eyes, he will still, if he has binocular vision, be able to read every word, because the letters that are hidden from the left eye will be seen by the right, and *vice versa.* If he has not binocular vision, the pencil obviously will cut out part of what he is reading. Hence, this simple test enables us to tell whether or not he is using one or both eyes.

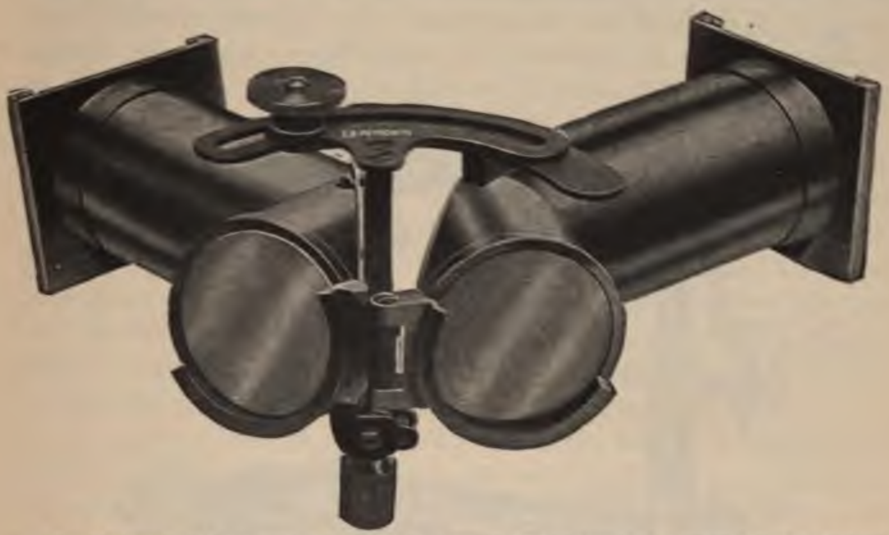


FIG. 8.—WORTH'S AMBLYOSCOPE, MODIFIED BY BLACK.—The amblyoscope consists of two tubes, bent and joined so as to admit of being converged and diverged through a considerable angle. In the Black modification here shown, one of the tubes can also be raised or lowered, an arrangement advantageous when the two eyes are not on the same level. My own model is also fitted with a graduated arc which shows the amount of the deviation or of the convergence and divergence employed. The further end of each tube carries a picture mounted on a translucent plate of celluloid. A mirror at the bent portion of the tube enables the patient looking through the eyepiece to see the picture with the corresponding eye. If he has binocular vision he will see two pictures, one with each eye, and by winging the tubes in or out he will get these two pictures together and fuse them.

6. *Stereoscope and Amblyoscope.*—A patient who has binocular vision will be able to see two dissimilar images placed in the stereoscope and to fuse them into a single image. This, of course, is impossible for one who has only monocular vision. Different forms of the stereoscope have been devised, both for determining the presence or absence

The Maddox rod can also be used to in [illegible]
torsion of the eye,—*i.e.*, any tilting of its [illegible]
or left. If there is, the streak of light [illegible]
is set directly vertical, and the number [illegible]
rod has to be rotated in order to make [illegible]
will indicate the amount of the torsion [illegible]
of the right eye is rotated 10° toward [illegible]
the Maddox rod out 10° in order [illegible]
vertical.

4. *Phorometer.*—The phoro [illegible]
artificial diplopia by means of [illegible]
double images deducing whether [illegible]
not.

Stevens's phorometer con [illegible]
that they can be rotated sim [illegible]
prisms are first set so tha [illegible]
then looking through the [illegible]
that of the right eye be [illegible]

[illegible] rough l
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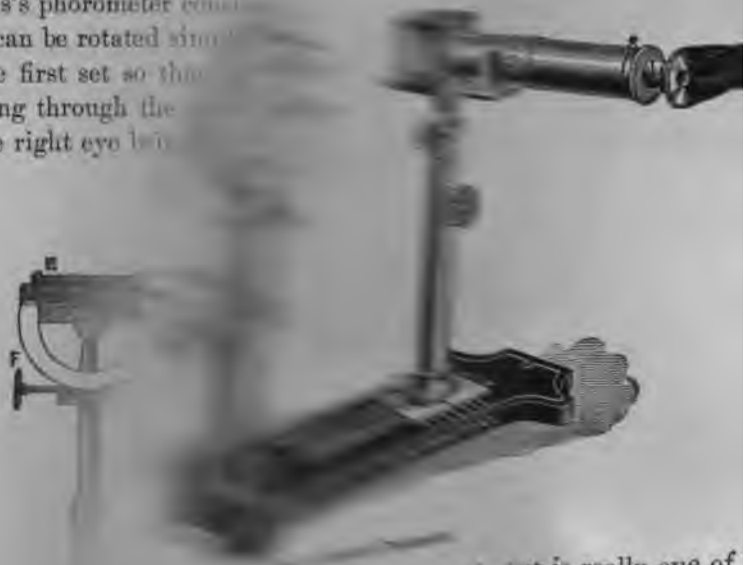

[illegible] the test is really one of

[illegible] each eye separately, and
[illegible] can be determined by the
[illegible] With the latter the patient
the image [illegible] position is fixing the zero mark
right imag [illegible] a pair of dots set very close
In ca [illegible] perimeter arc and the patient di-
they are [illegible] not his head. The moment he
index. [illegible] that the eye has reached the limit
To [illegible] direction,—the two dots will blur and
right-ha [illegible] Then the point to which the test-object

has been carried on the perimeter arc shows the amount in degrees that the eye has rotated in the given direction.

By ascertaining the limits of the excursion in different meridians the field of fixation may be plotted out on a perimetric chart in the same way as is done for the field of vision.

V. **Tests of Binocular Movements.**—1. *Tests of Parallel Movements.* —There are two ways in which the ability of the eyes to perform parallel movements can be tested.

First, we may ascertain the *field of binocular fixation,*—that is, we may find how far the two eyes can move in any given direction and keep pace with each other in their movements, so that they both fix the same object. The usual way of doing this is to make the two eyes follow a pencil or similar object in the different directions of the gaze and note whether either eye lags or falls behind in making the movement. This lagging can usually be detected pretty readily when there is a deficiency of either inward or outward rotation. It is well marked, therefore, in insufficiency or paresis of the external and internal recti. It is much more difficult to tell whether either eye lags behind the other when the two are moving up or down. This is especially the case when the eyes are directed up and to the right or up and to the left, for then the outward eye usually appears to be the lower, even when it can be readily proved that both are accurately fixing the same object.

A more accurate means of determining the field of binocular fixation is by making the *screen test* in the various directions of the gaze. A noticeable deviation behind the screen would show that there was at least a tendency to deflection of the eyes. If this deviation was greater than in the primary position it would show the deflection was increasing as the eyes were carried away from the latter; and if the method of binocular uncovering (page 199) should show that there was a squint in one of the outlying positions which did not exist in the primary position, it would indicate that the deflection had become so great that one eye could no longer keep pace with the other and perform fixation with it.

Second, we may determine the *field of binocular single vision.* In doing this we place a red glass before the patient's right eye and stand three feet in front of him with a light which we pass to the right, to the left, up and to the right, up and to the left, down and to the right, and down and to the left. We direct him to follow the light with his eyes without moving his head, and if in any direction of the gaze he gets double vision, we ask him how the images are related to each other, how far they are apart, and whether one or the other is tilted or not.

Normally, the patient should be able to carry the gaze through a range of 40° at least in any given direction without seeing double, and most people, indeed, will not see double at all, no matter how far the test-object is carried.

Diplopia developing within 30° of the primary position is distinctly pathological, and indicates insufficiency of some of the ocular muscles.

It is sometimes important to determine the *field of binocular single vision for distance.* In this case the patient is made to keep his eyes fixed on a small brilliant light fifteen or twenty feet off while he turns his head in various directions. A diplopia developed when the head is turned to the left, etc., is really a diplopia produced when the eyes are turned to the right, etc. (The method of taking and recording is described more fully in Section II. under Diagnosis of Paralysis.)

In taking the field of binocular single vision we may at the same time take the *field of binocular fixation,* for, as the patient follows the light with his eyes, we can, by watching closely, ascertain whether either eye seems to lag when the light is carried in any direction.

2. *Tests of Convergence.*—These are threefold: (*a*) By the *screen* and *diplopia tests* (Maddox rod, phorometer) we measure the amount of any deviation that may be present when the eyes are converged on a point some ten inches distant. If this deviation is much greater than that found for distance by the same tests, it argues some abnormality of the converging power, either in the way of excess or defect. Such a determination is of great importance and should form a regular part of the examination for muscular anomalies.

(*b*) We may determine the *convergence near-point,* or the nearest point to which the patient can converge his eyes by the utmost effort. This should be not more than two inches from the root of the nose. If more than this it argues an insufficiency of convergence.

Even when, as in divergent squint, the patient has no absolute convergence near-point, since he cannot produce binocular fixation at any distance, far or near, he yet often makes strong converging efforts, swinging the eyes nearly but not quite to meet the object of fixation as it is brought towards him. The point nearest to the eyes at which he gives up this effort, and allows the eyes to diverge widely, is called his *relative convergence near-point.*

(*c*) We may determine the *prism-convergence* (wrongly called the *adduction*). This is measured by the ability to overcome,—*i.e.*, see single with—prisms held, base out, before the eyes.

The amount of prism-convergence that a patient can do at the first examination will vary from 10° to 20° or more, but by a little practice most people can overcome prisms of 50°, 60°, or more, and this either when looking at a distant object or when looking at an object a foot away.

3. *Tests of Divergence.*—The ability of the eyes to diverge is measured by the prism, base in, that they can overcome. When looking at a distant object most eyes can overcome from 4° to 8° of prism, base in, this constituting the *prism-divergence* or so-called *abduction* for distance.

A prism-divergence of less than 4° in general indicates weak, and of over 8° or 9° an excessive, divergence power.

How the Tests are made in Practice.—In examining a case for a motor anomaly of the eye we proceed, as follows:

A. We first determine the *relations for distance* by placing the test-object far off,—if possible, twenty feet or more,—from the eyes and then measuring the amount of deviation by

(1) The *screen* and (2) the *parallax* simultaneously. At the same time we determine by the method described on page 21 whether the deviation is a squint or a heterophoria.

(3) The *Maddox rod*, testing both for hyperphoria and for lateral deviation.

(4) The *phorometer*, testing both for hyperphoria and for lateral deviation.

B. We measure the *deviation for near points* (with the test-object held at about ten inches) by

(5) The *phorometer;* (6) the *screen* and (7) the *parallax* simultaneously; (2) the *Maddox rod.*

In this case, again, we ascertain by the screen test whether the deviation is a squint or a heterophoria.

C. We determine:

(9) The *convergence near-point.*

(10) The *prism-divergence*, with prisms held, base in, before the eyes, the test object in this case being at twenty feet.

(11) The *prism-convergence* by means of prisms held, base out, before the eyes, the test object again being at twenty feet.

(12) The *field of binocular single vision*, by using as the test object a candle held at three feet from the eye. In making this test we note at the same time whether the two eyes follow the candle in all directions, or whether one eye lags behind the other in performing some particular movement,—that is, we determine

(13) The *field of binocular fixation.*

Other tests may be employed, but these are sufficient for all practical purposes. Indeed, in all ordinary cases we may omit the use of the phorometer, and, provided the convergence near-point is normal, we may also dispense with the determination of the prism-convergence.

What the Test shows in Normal Cases.—With the test made in the way indicated above, the eye-muscles may be regarded as normal if

(*a*) Tests 1 and 6 show that there is no squint, and Tests 1 to 8 show that there is only a minimal amount of heterophoria (less than 2° of esophoria or exophoria [*] and less than ½° of hyperphoria) for both distance and near.

(*b*) The convergence near-point is less than two inches from the root of the nose.

(*c*) Prism-divergence is not less than 4° and not over 8° (prism = 2° to 4° of actual deviation).

(*d*) Prism-convergence after a few trials can be run up to 15° or 20° or more.[†]

(*e*) Tests 12 and 13 show that the fields of binocular fixation and binocular single vision are normal,—*i.e.*, the patient having a red glass before one eye and following a light that is carried in all possible directions sees everywhere a single particolored light, and each eye moves equally with its fellow without lagging anywhere.

Anomalies of Eye-Muscles and Eye Movements; Etiological Classification of Deviations.—The various tests just outlined enable us to make a diagnosis of the ordinary motor anomalies of the eye commonly met with. In considering these anomalies we must distinguish between—

A. Deviations due to anomalies of one or more of the ocular muscles; deviations, that is, which are produced by some affection of the muscles themselves, of the nerves supplying them, or of the nerve nuclei.

B. Deviations due to derangement of one of the co-ordinating movements of the eyes. The muscles themselves in this case and their central connections up to and including the nuclei are intact, but the supranuclear or internuclear nervous mechanism presiding over the functions of convergence, divergence, and associated parallel movements is in some way out of gear.

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[*] The Maddox rod often shows an excess of esophoria for distance (1–2° more than the other tests), and the phorometer sometimes shows 2–3° of exophoria for near when the other tests prove that there is orthophoria.

[†] There is no absolute or fixed relation between the amount of prism-divergence (abduction) and prism-convergence (adduction). The former amounting to from 5° to 8°, cannot, as a rule, be altered in any given case by exercise. Prism-convergence, on the other hand, may range anywhere from 12° to 30° at the first trial, and, however low at the outset, can almost always by practice be brought up to 60° or 70° or even more.

We have, therefore, to consider in succession—

A. Anomalies of the ocular muscles:
  (1) Paralysis of the ocular muscles.
  (2) Spasm of the ocular muscles.
B. Anomalies of the co-ordinate movements:
  (1) Anomalies of convergence:
    Convergence-excess.
    Convergence-insufficiency and convergence-paralysis.
  (2) Anomalies of divergence:
    Divergence-excess.
    Divergence-insufficiency and divergence-paralysis.
  (3) Anomalies of parallel movements:
    Conjugate paralysis.
    Conjugate spasm.
    Nystagmus.

SECTION II.

PARALYSIS OF THE INDIVIDUAL MUSCLES.

Symptoms.—A. Diplopia.—All the subjective symptoms of paralysis are due chiefly to the diplopia that the paralysis causes. Since this diplopia itself is due to the fact that the eye, weakened by paralysis, fails to keep pace with its fellow, and since this diplopia will become most apparent as soon as the affected muscle is called into action, it is evident that the diplopia, and with it the other symptoms, will increase when the eyes are carried into the field of action of the paralyzed muscle. For example, in paralysis of the right abducens, diplopia will increase fast when the eyes are directed to the right; whereas, when the eyes are directed to the left, the diplopia will diminish and ultimately disappear, because in that direction of the gaze the affected muscle takes no part in moving the eye.

The characteristic mark of paralysis, therefore, is a diplopia which increases progressively and fast as the eyes are carried in some definite direction and diminishes when the eyes are carried in the contrary way.

The kind of diplopia present in the various forms of paralysis will be given below under the head of diagnosis.

The other symptoms beside diplopia which are found in paralysis are as follows:

B. False Projection.—In a fresh case of paralysis of the right abducens, the patient when told to close his left eye and quickly put his finger on an object situated to his right, will in general shoot too far to the right of the object; in other words, the object appears to him to be further to the right than it really is. This is due to the fact that in order to look at the object with the paretic eye he has to exert an excessive amount of nerve energy, such as corresponds in his former experience to a marked movement of the eye to the right—that is, he has to work hard to move his eye a little and he thinks, therefore, that he really moved it a great deal.

So, too, in paralysis of a left rotator, the patient will project the object too far to the left; in paralysis of an elevator he will project it too high up, and in paralysis of a depressor too low down.

C. Apparent Movement of Objects *pseudokinesis*).—When a patient with paralysis of the right abducens looks with his paralyzed eye at an object situated to the left, the latter will appear in its proper place; if, then, he suddenly turns his head to the left, so that the object is now somewhat to the right, it will no longer appear in its proper place, but somewhat to the right of it. In other words, it will seem to make a sudden jump from left to right. And, in general, when the eye is carried in a direction corresponding to the field of action of a paralyzed muscle, objects at which it is looking will suddenly move, the *direction of the movement being always that in which the muscle itself would naturally turn the eye.* That is, in paralysis of a right rotator objects will appear to go to the right, in paralysis of a left rotator to the left, in paralysis of an elevator they will rise up and in paralysis of a depressor they will go down.

D. Vertigo.—The changing diplopia and the apparent movement of objects seen with the paralyzed eye produce vertigo, which is often a very marked and distressing symptom of paralysis.

E. Attitude of Head.—All the symptoms above described increase in proportion as the eyes are carried in the special direction in which the muscle, if it were not paralyzed, would move the eye. To avoid them, the patient instinctively *turns his head in this direction* so that his eyes shall be turned in the opposite way. Thus, in paralysis of a right rotator (right externus, left internus), he will turn his head to the right, so that even when looking at objects straight in front of him, the eyes themselves will be directed to the left. Similarly in paralysis of a left rotator, he will turn his head to the left. In paralysis of an elevator, he may throw his head up and back, and in paralysis of a depressor, he may throw it forward and down. In paralysis of an elevator or de-

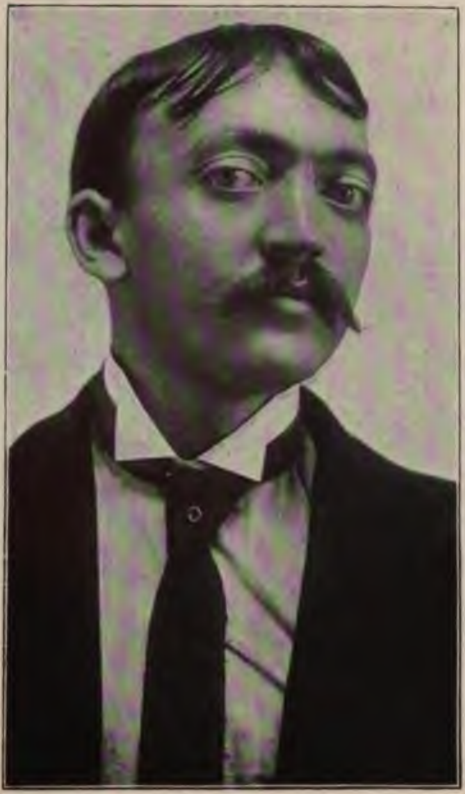

Fig. 10.

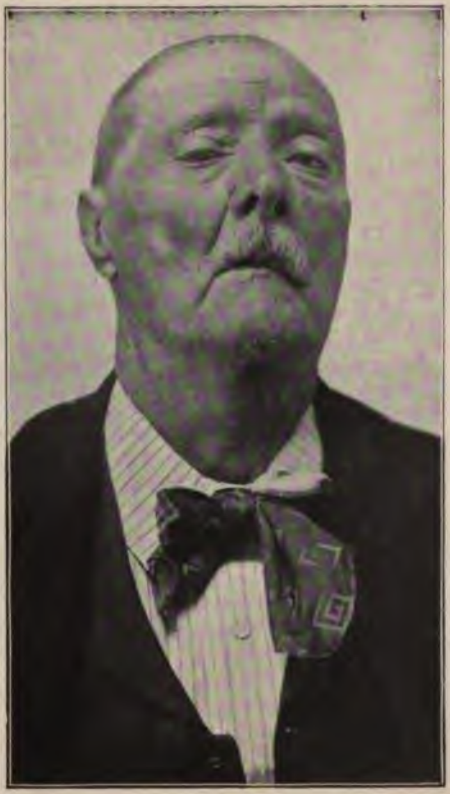

Fig. 11.

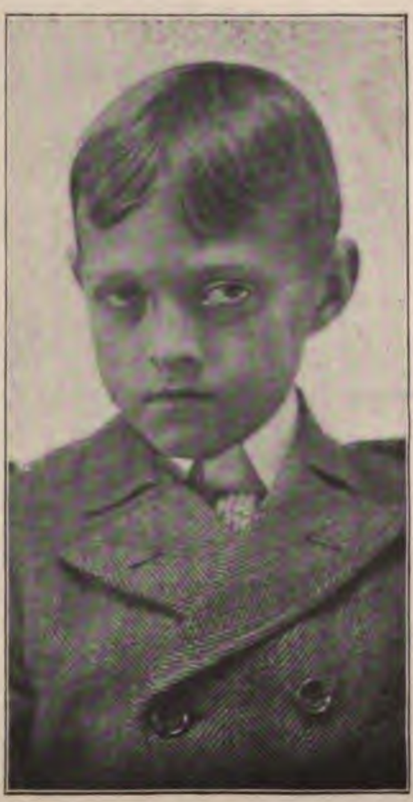

Fig. 12.

Figures illustrating compensatory positions of the head in paralysis of eye muscles. Fig. 10 shows position assumed when the field of diplopia is to the left (paralysis of a left rotator—L. externus, R. internus); Fig. 11, the position when the field of diplopia is up and to the left (paralysis of a left-hand elevator—L. superior rectus, R. inferior oblique); Fig. 12, the position when the field of diplopia is down and to the right (paralysis of a right-hand depressor—R. inferior rectus, L. superior oblique).—W. T. Shoemaker in International Clinics, Vol. IV., 12th Series.

pressor, however, the easiest and most usual way of obviating the diplopia is for the patient to tilt the head towards one shoulder. This depresses the corresponding image,* and thus enables the patient to bring the images on a level in case there is vertical diplopia. The head at the same time is usually turned somewhat to right or left (Fig. 13).

This constant maintenance of the head in a constrained position, to obviate the diplopia due to a paralysis, has, in some instances, *produced torticollis.* (See cases cited by Posey, Section on Ophthalmology, Fifty-third Annual Meeting of the American Medical Association.)

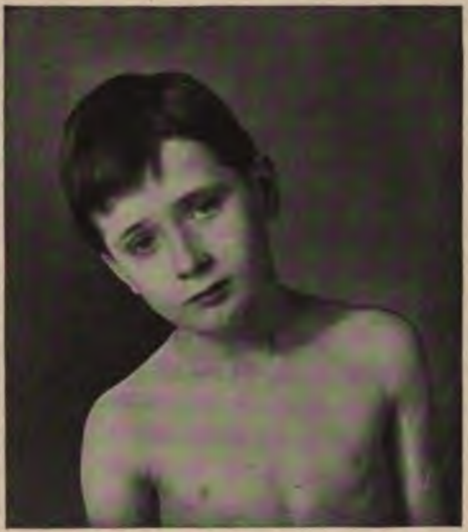

FIG. 13.—Showing torticollis from vertical deviation of one eye. Case of Dr. Posey

F. Other Symptoms.—In paralysis we often meet with other symptoms, such as headache, various forms of anæsthesia, paræsthesia, etc. The headache may occasionally be a reflex symptom, due to the confusion and strain imposed by the paralysis, but this seems to be very rarely the case. Generally speaking, the headache and other symptoms are the result of the cause producing the paralysis and not of the paralysis itself.

Diagnosis of Paralysis.—In diagnosticating paralysis we apply the various tests already described:

A. Deviation in Primary Position.—The screen-test and the different diplopia tests (parallax, Maddox rod, and phorometer) show the presence of a deviation even in the primary position. Thus, in paralysis of the abducens, the eyes will be unduly converged so that all the tests will show a pronounced esophoria; in paralysis of the internus there will be exophoria; and in paralysis of the elevators or depressors, hyperphoria.

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* That is, if he tilts the head to the right shoulder, the right-hand image, whether belonging to the right or to the left eye, is carried down.

In applying the screen-test, we will generally notice that the deviation of the paralyzed eye behind the screen (*primary deviation*) is less than that of the sound eye (*secondary deviation*). The reason for this is as follows: Suppose that the right abducens is paralyzed and that this eye, in consequence when screened, deviates inward 1 millimetre; when the screen is transferred to the left eye the right will have to turn out 1 millimetre in order to fix. To do this, the eye must employ its paralyzed externus and, as this is an inefficient instrument, must exert a great deal of nerve power. This same nervous force is necessarily conveyed to the left eye at the same time, impelling it to turn very strongly to the right. As the muscles of the left eye are intact, it is able to obey this impulse fully, and hence moves to the right, not one but several millimetres.

The difference between the primary and the secondary deviation is best measured with prisms. If, for example, a prism of 10°, before the right eye will exactly abrogate the screen deviation, but it takes a prism of 20° before the left eye to do the same thing, we know that the secondary deviation of the left eye is about twice as great as the primary deviation of the right.

B. Limitation of Movement.—The tests for the *monocular field of fixation* made with the tropometer or perimeter show the limitation of movement of the affected eye in some particular direction of the gaze. This test, however, is often unsatisfactory and is better replaced by the following tests for determining the field of binocular fixation and binocular single vision.

C. Lagging of One Eye behind the Other.—The *field of binocular fixation* is determined in the way already described (page 205). In a case of paralysis we find that as the two eyes follow a moving object, one lags markedly behind the other as soon as the eye is carried in the direction of the field of action of the paralyzed muscle. Thus, in paralysis of the right abducens the right eye will begin to falter and fall behind as soon as the eyes are turned to the right; in paralysis of an elevator, the affected eye will fail to rise with its fellow, and in paralysis of a depressor, it will not go down as well.

By applying the *screen-test* in the different directions of the gaze, it may be possible to show that there is binocular fixation when the eyes are turned in one way, say the right, and that there is a marked deviation from binocular fixation when the eyes are turned in the opposite way, to the left. This will be evidence of the paresis of a left rotator.

D. Diplopia.—By far the most satisfactory evidence of paralysis is that afforded by the determination of the *field of binocular single vision,* —*i.e.,* by ascertaining the amount and kind of diplopia present in the different directions of the gaze.

The *characteristic diplopia* found in paralysis of each muscle is as follows:

In paralysis of the *right abducens* there is homonymous diplopia, increasing fast to the right. In paralysis of the *left abducens* there is homonymous diplopia which increases fast to the left. In both cases the diplopia will be slightly less when the eyes are directed up and slightly greater when the eyes are directed down.

In paralysis of the *right internal rectus* there will be crossed diplopia increasing fast to the left. In paralysis of the *left internus* there will be crossed diplopia increasing fast to the right. In both cases the crossed diplopia will increase somewhat when the eyes are turned up and diminish somewhat when they are turned down.

In paralysis of the *right superior rectus* there will be vertical diplopia, with the image of the right eye higher (left diplopia). This will increase as the eyes are carried up, and particularly as they are carried up and to the right. In general, but not invariably, there will be also a moderate crossed diplopia, which will increase in looking to the left. The image of the right eye will be tilted to the left.

In paralysis of the *left superior rectus* there will be a vertical diplopia (right diplopia), increasing up and to the left, and a crossed diplopia, increasing up and to the right. The image of the left eye will be tilted to the right.

In paralysis of the *right inferior rectus* there will be a vertical (right) diplopia, increasing as the eyes are carried down and to the right, and usually, but not always, a crossed diplopia, which will increase in looking down and to the left. The image of the right eye will be tilted to the right.

In paralysis of the *left inferior rectus* there will be a vertical (left) diplopia, increasing down and to the left, while the crossed diplopia, if present, will increase in looking down and to the right. The image of the left eye will be tilted to the left.

In paralysis of the *right superior oblique* there will be a vertical (right) diplopia, which will increase as the eyes are directed down and to the left, and, usually, also an homonymous diplopia, which will increase down and to the right. The image of the right eye will be tilted to the left.

In paralysis of the *left superior oblique* there will be a left diplopia, increasing down and to the right and an homonymous diplopia, increasing down and to the left. The image of the left eye will be tilted to the right.

In paralysis of the *right inferior oblique* there will be a left diplopia, which will increase as the eyes are carried up and to the left, and sometimes, but not always, an homonymous diplopia, which will increase as the eyes are carried up and to the right. The image of the right eye will be tilted to the right.

In paralysis of the *left inferior oblique* there will be a right diplopia, increasing up and to the right, and an homonymous diplopia, increasing up and to the left. The image of the left eye will be tilted to the left.

We meet with various combined paralyses producing more or less complicated forms of diplopia. Chief among these is the *oculo-motor paralysis,* or paralysis of all the muscles supplied by the third nerve. In this case the eye can be moved outward by the abducens, but it cannot be moved in any other way, for, although the superior oblique is still acting, it cannot move the eye downward since it is not efficient as a depressor when the eye is in a position of abduction, as it is in paralysis of this sort. The most that the superior oblique can do under these conditions is to give the eye a slight inward rotation upon its antero-posterior axis when the patient attempts to turn the eye down.

In a right oculo-motor paralysis there will be a crossed diplopia, due to the relative divergence of the paralyzed eye, and usually also a slight left diplopia, because the eye sags somewhat below the level of the other. This left diplopia will increase fast as the eyes are carried up. On the other hand, when the eyes are carried down, the left diplopia will be transformed into a vertical diplopia of the opposite kind (right), which will increase fast as the eyes are depressed.

The diagnosis of a paralysis from the behavior of the double images will be facilitated by the following table:

TABLE SHOWING FROM DIPLOPIA WHICH MUSCLE IS PARALYZED.

DH=homonymous diplopia.
DX=crossed diplopia.
DR=right diplopia (vertical diplopia with image of right eye below).
DL=left diplopia (vertical diplopia with image of left eye below).
Er, El, Eu, Ed, Eu and r, etc.=respectively "when both eyes are directed to the "*right*" "to the *left*," "*up*," "*down*," "*up and to the right*," etc.
>>=increasing fast and progressively.

A. The diplopia is lateral (DX or DH) >> in Er or El. (= paralysis of a lateral rotator.)

Diplopia >> in Er. (=paralysis of a RIGHT ROTATOR.)	Diplopia >> in El. (=paralysis of a LEFT ROTATOR.)
DH >> in Er. *R. externus.*	DH >> in El. *L. externus.*
DX >> in Er. *L. internus.*	DX >> in El. *R. internus.*

B. The diplopia is vertical (DR or DL) >> in Eu. (=paralysis of an elevator.)

Diplopia >> in Er (Eu and r). (=paralysis of a RIGHT-HAND ELEVATOR.)	Diplopia >> in El (Eu and l). (=paralysis of a LEFT-HAND ELEVATOR).
DL >> in Eu and r. *R. superior rectus.*	DR >> in Eu and l. *L. superior rectus.*
DR >> in Eu and r. *L. inferior oblique.*	DL >> in Eu and l. *R. inferior oblique.*

C. The diplopia is vertical (DR or DL) >> in Ed. (=paralysis of a depressor.)

Diplopia >> in Er (Ed and r). (=paralysis of a RIGHT-HAND DEPRESSOR.)	Diplopia >> in El (Ed and l). (=paralysis of a LEFT-HAND DEPRESSOR.)
DR >> in Ed and r. *R. inferior rectus.*	DL >> in Ed and l. *L. inferior rectus.*
DL >> in Ed and r. *L. superior oblique.*	DR >> in Ed and l. *R. superior oblique.*

A good rule to enable us to determine which is the paralyzed eye is the following:

When, in moving the eyes in a given direction, the double images begin to separate, that image which moves the faster and the further belongs to the paralyzed eye.

And a rule to determine the kind of double vision produced by a given paralysis is,—*the image belonging to the paralyzed eye is displaced in just that direction in which the affected muscle, if intact, would naturally move the eye.* Thus, in paralysis of the right superior rectus, which moves the right eye up and to the left and tilts the vertical meridian to the left, the image of the right eye is up (higher), deflected to the left (crossed diplopia), and tilted to the left.

The double images may be conveniently *plotted on a diagram,* as shown in Fig. 14.

Fixation with the Paralyzed Eye.—A marked change in the appearances presented, although not in the actual conditions present, is produced by fixation with the paralyzed eye. The paretic eye, in this case, is forced into position, and the sound eye deviates. Now the force applied to the sound eye to make it deviate is equal to the force applied to the paretic eye to make it get into position. This latter force, however, must be very considerable, and the more so the greater the degree of paresis. Hence, as we have seen (p. 212), the deviation of the sound eye when the paretic eye fixes (secondary deviation) is much greater than the deviation of the paretic eye when the sound eye fixes (primary deviation). Accordingly, the effect of fixation with the paralyzed eye is to *increase the deviation* sometimes to double or treble of its original nt. The result is that the double images are put much further

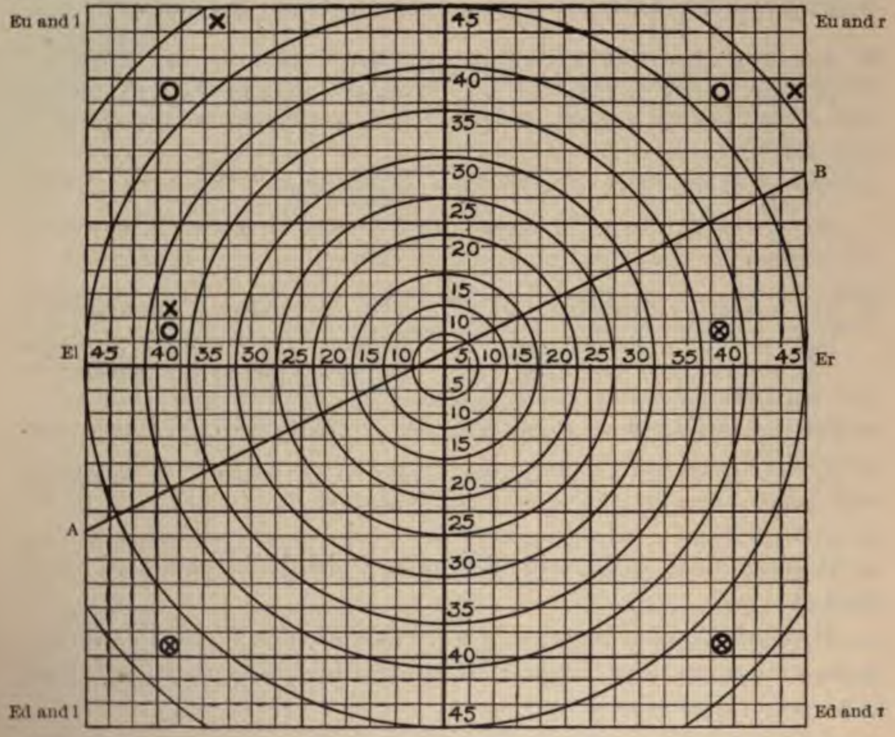

FIG. 14.—DIAGRAM FOR PLOTTING DOUBLE IMAGES IN PARALYSIS.—The diagram is made on the assumption that the field of binocular single vision is taken at a distance of thirty inches and projected on a tangent plane at that distance (represented by the large square). The fine cross lines denote intervals of 2 inches; the heavier cross lines, intervals of one foot. The concentric circles denote intervals of 5° plotted on the tangent plane. The diagram is used as follows: The light is held thirty inches from the patient and carried into the six cardinal positions "Eyes right" (Er), "Eyes up and right" (Eu and r), etc., and the amount of diplopia, vertical and lateral, in inches is laid off on the diagram. In the case shown (one of paralysis of the left superior rectus), in which the image of the right eye is denoted by a circle and that of the left by a cross, the vertical distance between the two images when the eyes are directed two feet up and two feet to the left is six inches and the lateral distance is four inches. The diagram shows that this means that when the eyes are elevated 38° and also carried 38° to the left, the vertical diplopia is over 5° and the lateral diplopia about 3° of arc. When the eyes are directed the same distance up and to the right there is simple crossed diplopia of 5°, and if directed two feet straight to the left a simple vertical diplopia of 2.5°. Below the oblique line AB (that is in the positions Er, Ed and r, and Ed and l) there is single vision. The diagram can be used for any other distance than thirty inches, provided the proper allowance is made. Thus if the light were held five feet from the patient, the fine cross lines would represent intervals of four inches, and the heavy cross lines intervals of two feet.

apart, and hence interfere less with each other, cause less confusion, and are more readily ignored. This, in fact, as Graefe says, is evidently the reason why the patient adopts this manœuvre at all.

This secondary deviation of the sound eye, when the paretic eye fixes, always appears under the *guise of a spasm of the associate* (page 17) of the paralyzed muscle. Thus, if the right superior rectus is paralyzed, and yet this eye is used for fixation, the appearances produced will be those of a spasm of the left inferior oblique. For, in this case the right eye tends all the time to drop and can be kept in place only by an excessive nervous effort. This effort will be very great when the eyes are directed to the right, because in this position its paretic superior rectus is the only muscle that lifts the right eye. On the other hand, when the eyes are directed to the left, the right superior rectus acts very little as an elevator, and, as the muscle (the inferior oblique) which here does the main part of the work is normal, but little extra effort will be required to keep the eye in place. Now the extra effort that is put forth to keep the right eye in place is exerted equally upon the left eye. Hence, when looking to the right the left eye will rise unduly (shoot obliquely up), while in looking to the left, it will rise little or not at all above the proper level. This is just the effect that would be produced by a spasm of the right-hand elevator (inferior oblique) of the left eye. Similarly, as shown in the table on page 246, a paralysis of the right superior oblique will, if the right eye is fixing, give the appearance of a spasm of the left inferior rectus, etc.

Fixation with the paralyzed eye is not infrequent. It is especially apt to occur when the paretic eye has the better vision, although it may also occur when this is the poorer eye of the two. It seems to be comparatively frequent in paralysis of the superior rectus, and not very frequent in abducens paralysis.

Sometimes the patient fixes *alternately with the paretic and the sound eye.* Thus, in one case that I saw, a boy with a paresis of the left superior rectus, either fixed with the right eye so as to have a left hypotropia, or with the left eye so as to have a right hypertropia (alternating vertical squint). In the same way an alternating convergent squint may be produced by alternate fixation in a case of paralysis of one abducens.

Varieties of Paralysis.—Isolated and Combined Paralyses.—Paralysis may affect one or several ocular muscles.

Isolated paralysis of the *abducens* is very common and such paralysis is occasionally bilateral.

Oculomotor paralysis, complete or partial, is the next most frequent form, and all sorts of combinations occur. In complete oculomotor paralysis, not only are the motions of the eye affected, as stated (page 39), but there are also ptosis (or drooping of the lid from paralysis of the levator), paretic mydriasis, and paralysis of accommodation. Frequently the accommodation and the iris alone are paralyzed (*ophthalmoplegia interior* or *interna*) (see page 312); in other cases just these muscles are exempt. In other cases still, *ptosis* alone is present and is often bilateral. In ptosis not only is there complete or partial drooping of the lid, but there is also considerable wrinkling of the brow, because the patient endeavors by compensatory contraction of the frontalis to make up for the deficient action of the levator.

The next most frequent form of paralysis in the author's experience (differing therein from that of other observers) is isolated *paralysis of the superior rectus.* It also is sometimes bilateral.

Next in order comes paralysis of the *superior oblique* (trochlear paralysis). This is fairly common, but it is very rarely indeed bilateral, only two or three such cases being recorded, one of which occurred in the author's own experience.

Isolated paralysis of the *inferior rectus* is fairly frequent, occurring in the author's experience more often than that of the *internal rectus.*

Isolated paralysis of the *inferior oblique* is very uncommon. Some thirty cases have been recorded, but less than half of these are authentic. Yet it is probably more frequent than has been supposed, since the author has seen no less than four cases, all incomplete. One of these was congenital. In a single instance, reported by Bielschowsky, the paralysis was bilateral.

In some cases there is total paralysis of all the muscles moving the eyeball and of the interior muscles as well (*total ophthalmoplegia*). The eyeball is then perfectly motionless, the pupil widely dilated and the accommodation entirely absent. More or less complete paralysis of all the exterior muscles with exemption of the sphincter and ciliary muscles, is called *ophthalmoplegia exterior* or *externa.*

Etiology.—Etiologically, we have to distinguish three varieties of paralysis,—structural, insertional, and innervational.

Structural paralysis is due to some abnormality of the muscle itself. Such abnormality may be the result of congenital defect, solution of continuity, inflammation of the muscle or its surroundings, atrophy, etc. (For fuller details see section on Orbital Paralysis.)

Insertional paralysis is the form in which the muscle and its supplying nerve are normal, but the muscle is improperly inserted, either as the result of congenital misplacement, accident, or operation. (See for fuller details Orbital Paralysis.)

Innervational paralysis is the form in which the muscle and tendon are normal, but there is some abnormality of the nerve or its central connections. Such abnormality may consist in (*a*) congenital deficiency; (*b*) solution of continuity; (*c*) anæmia or hyperæmia, due either to general disturbance of the cerebral circulation, or to pressure upon the vessels supplying the nerve or its nucleus; (*d*) atrophy of the nerve or its nucleus, due to pressure of exudates, inflammatory swellings, callus, hemorrhages, aneurismal, atheromatous or thrombotic vessels, or neoplasms; (*e*) softening or degeneration of the nerve or its nucleus.

With regard to *site* we divide paralysis into extracranial and intracranial.

Extracranial (Orbital or Peripheral) Paralysis.—In a certain number of cases orbital paralysis is *congenital.* (For varieties see below under Exciting Cause, Congenital Conditions.)

Orbital paralysis (or insufficiency) may be due to *developmental changes* in the muscles or nerves. One variety of this is the relative insufficiency of the interni and preponderance of the externi, caused by the gradual separation of the orbits, which takes place in the development of the skull in childhood. This separation, as Weiss showed, by giving the externi a larger and the interni a smaller arc of contact with the eyeball, tends to produce a relative divergence of the eyes.

Some orbital paralyses are due to *traumatism,* either from the direct effect of the injury upon the muscle or tendon, or the remote effects produced by the pressure of effused blood and callus in the case of fractures, or to cicatricial contraction in the tissues of the orbit. (See for further particulars Exciting Causes, Traumatism.)

Orbital paralysis may, very rarely, be due to *disease of the muscles* themselves, as to myositis, simple (Gleason) or syphilitic (Hochheim); to hyaline degeneration (Nuel); to degenerative changes occurring in progressive muscular dystrophy (Groenouw, Uhthoff) and in acute ascending paralysis (Goebel); and to metastatic tumors (six cases cited by Groenouw and Bernheimer).

In other cases it is produced by *disease of the nerves,* especially a primary tabic degeneration (Dejerine, Marina); very rarely by secondary new growths occurring in the nerves (Bernheimer).

A more usual cause of orbital paralysis is the presence of *tumors* (especially gummata, cavernous growths, exostoses, and sarcomata) or *inflammatory processes* (orbital cellulitis, tenonitis, periostitis, mucocele) of the contents, walls, and accessory cavities of the orbit. These processes may act by causing secondary changes (inflammation, degeneration, or atrophy) in the muscles and nerves, in which case the picture presented will be that of a true paralysis of some muscle or group of muscles. They often, however, act in a *purely mechanical way* by pressing the eyeball forward or by enveloping it and its muscles in rigid masses preventing movement. In this case the deviation of the eye, the restriction of movement, and the diplopia produced may differ altogether from what we find in any of the types of paralysis we have described.

A similar mechanical restriction of eye movements with more or less aberrant diplopia may be caused by a pterygium or pseudopterygium.

Intracranial Paralysis comprises basal, nuclear, and cortical paralysis.

Basal (and Foraminal) Paralysis.—By *basal* paralysis we understand a paralysis due to lesion of the nerve-trunk after its emergence from the brain and before its exit from the skull; by a *foraminal* paralysis, one due to lesion of the nerve as it passes through the sphenoidal fissure. The nerves in traversing this fissure pass through veritable foramina, whose walls are made up of rigid fascia. This arrangement, while protecting them from injury, renders them more liable to compression and strangulation.

Basal and foraminal paralyses are due to—

(1) *Arrested or imperfect development* of the nerve-trunks (congenital paralysis). Very rare.

(2) *Solution of continuity* of the nerve from blows on the head causing fractures of the base, or from shot or stab wounds of the mouth or orbit.

(3) *Hemorrhage into the nerves* from traumatism, or from the spontaneous rupture of aneurismal or sclerotic vessels.

(4) *Inflammation* or *degeneration* of the nerves from syphilis or other causes.

(5) *Tumors* of the nerves.

(6) *Pressure on the nerves* from (*a*) hemorrhage; (*b*) inflammatory exudate (meningitis, periostitis) including callus; (*c*) hard or distended vessels; (*d*) tumors (especially gummata); and (*e*) general distention of the brain.

Nuclear and Fascicular Paralyses.—By *nuclear* paralysis we understand a paralysis due to lesion of the nerve-nucleus itself; by *fascicular* paralysis, one due to lesion of the root-fibres which emanate from the nucleus and which, before leaving the brain, coalesce to form the nerve-trunk.

Nuclear and fascicular paralyses are due to—

(1) Aplasia of the nucleus (*congenital paralysis*). Very rare. (It must be noted that, as Wilbrand and Saenger point out, some cases of congenital nuclear paralysis are due not to aplasia but to degeneration and atrophy of the nucleus from fetal or infantile disease. The distinction between the two would be that in aplasia there would not be, and in acquired atrophy there would be, a secondary denegeration of the nerve-trunk and muscles). (Compare Chapter XIII, Sect. II.)

(2) *Atrophy and degeneration* of the nucleus and root-fibres from syphilis, tabes, multiple sclerosis, and rarely from other cerebro-spinal diseases; also from poisons and the toxins of diseases like diphtheria, influenza, etc. (Compare Chapters XII and XV).

(3) *Anœmia, hyperœmia,* and *hemorrhage,* due usually to vascular changes (arteriosclerosis, endarteritis, miliary aneurisms), which are themselves generally the results of syphilis or the action of poisons (lead, diphtheria, influenza, etc.). A peculiar form of hemorrhagic disease of the nuclei and root-fibres is polioencephalitis superior (see below under Exciting Causes). Nuclear hemorrhages may also be due to traumatism.

(4) *Tumors,* especially gumma, more rarely glioma, sarcoma, and tuberculoma (Compare Chapter IX).

It must be noted that owing to the connection between the abducens nucleus of one side and the opposite oculo-motor nerve, a lesion (tumor, etc.) of the pons involving, say, the right abducens nucleus, does not cause simple paralysis of the right externus, but a conjugate paralysis (paralysis of dextroversion in both eyes). To produce simple paralysis of the abducens, the lesion must be so situated as to cut off the emergent root-fibres of the nerve; in other words, it must be a fascicular paralysis.

Supranuclear and Cortical Paralyses.—Lesions affecting the connections of the nuclei with the cortex and the cortex itself do not in general cause paralysis of the individual ocular muscles, but paralysis of the co-ordinated movements of the eye,—*i.e.,* of convergence, divergence, or the parallel movements. An exception seems to be ptosis, which in some twenty-five reported cases (Willbrand and Saenger) has been caused apparently by a cortical lesion and particularly one situated in the supramarginal and angular gyri (usually on the side opposite to the ptosis). Doubt, however, has been thrown on these cases, first, because often marked lesion of these parts may exist without causing ptosis; and, second, because even when ptosis is present we cannot always be sure that is not due to a remote effect (nuclear or basal lesion) produced by the cortical disease (Compare Chapter IX).

Exciting Causes of Paralysis.—The causes producing the various lesions enumerated above as present in the different forms of paralysis, orbital, basal, and nuclear, are as follows:

1. **Congenital Conditions.**—Congenital paralysis in most cases probably is due to *non-development* or *faulty development* of *the ocular muscles* themselves (orbital paralysis). The congenital malformations of the muscles causing paralysis are as follows:

(*a*) Absence of the muscle,—*e.g.,* to absence of the internus (Krause, Lawford), the externus (Krause, Bahr), the superior rectus (Seiler, Steinheim), both inferior recti (Stieren, A. E. Davis), and one or both obliques (Hartes, Seiler).

(*b*) Replacement of muscle by a more or less inelastic cord of connective tissue. This has been observed in the case of the internus (Uhthoff, Guende), and the externus (Baumgarten, Inouye, Alling, Axenfeld and Schürenberg, Evans, Bernheimer).

(*c*) Underdevelopment of the muscle. This has been observed by Lawford, Bach, and others. A moderate degree of underdevelopment is considered by Schneller to be a frequent cause of squint (*structural squint*). This view he bases on the findings in numerous squint operations. It is questionable, however, whether the underdevelopment that he found was not rather the result than the cause of the squint.

(*d*) Abnormal course and insertion of the muscle. Among the abnormal conditions found are: (1) Insertion of the muscle too far back, causing it to act as a retractor rather than a rotator of the eye (Heuck); (2) bifurcation of the tendon (Dieffenbach, Bahr *), and (3) the presence of adhesions or bands connecting the muscles with each other or with the orbital walls (Morgagni, Olbers and Wrisberg, Fitzgerald).

Much more rarely congenital paralysis is due to *aplasia of the nerves* (either in the orbit or the cranial cavity) or of their nuclei.

Some cases of congenital paralysis are *traumatic,* being produced either by direct pressure or by hemorrhage during delivery with the forceps. Such paralysis may affect the external rectus with or without involvement of the facial nerve, or the oculo-motor nerve, or the superior rectus with the levator (Groenouw, Nettleship).

Other rare cases of congenital paralysis are apparently due to *fetal*

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* In Bahr's case one of the slips was inserted too far back, causing the muscle to act partly as retractor.

inflammation or *degeneration* of the nerve nuclei (Wilbrand and Saenger).

Congenital paralyses may occur under all kinds of forms (Kunn, Zweig). The following comprise some of the more frequent types.

A. *Paralysis of the Superior Rectus.*—This is either unilateral or bilateral; partial or more often complete; and may or may not be associated with ptosis. This condition is quite frequent. It is probably due in most cases to underdevelopment or actual absence of the muscle.

B. *Ptosis.*—This is nearly always bilateral. It is usually incomplete, the lid simply drooping enough to give the patient a sleepy look. Sometimes, however, as in Lawford's cases, the eye is completely closed. Congenital ptosis is often combined with other defects, particularly with paralysis of the superior rectus.

It may be nuclear in origin, but is probably in most cases due to structural defects in the muscle itself (absence or conversion into connective tissue, underdevelopment, restriction by adhesions).

C. *Paralysis of the Externus with Paresis of the Internus and Retraction Movements.*—This condition usually presents itself under the guise of a well-marked syndrome characterized by some or all of the following features:

(1) The affected eye can move out little, if at all, beyond the median line.

(2) Its movement inward is more or less restricted. Moreover, even when the amount of movement inward is nearly normal, the movement itself is performed with difficulty, the eye always lagging behind its fellow, as shown by the presence of a crossed diplopia, increasing as the eye is turned in.

(3) When the affected eye is turned in, it retracts into the orbit and often also shoots obliquely up or down, and its palpebral fissure contracts, often considerably.

(4) The power of convergence is often considerably impaired.

(5) The field of single vision is very narrow indeed. The patient when he looks to the side of the affected eye has homonymous diplopia, which passes almost at once into a crossed diplopia when he begins looking in the opposite direction.

These cases are not uncommon, some fifty-five having been described (Duane). Usually the left eye, rarely both eyes, are affected.

The underlying cause of this condition is a congenital absence of the external rectus or its transformation into a connective tissue cord. In some cases this cord is elastic and the eye can be rotated inward by passive traction (with forceps); in other cases where the cord is inelastic this is not possible.

The retraction of the eyeball when adducted is due in most cases undoubtedly to the fact that the externus is thus inelastic, so that the eyeball, being hugged between an inextensible cord on the one side and the contracting internus on the other, must necessarily recede (Türk, Wolff). Other cases, however, are due to the fact that the internus is inserted too far back (Heuck, Evans), or has an additional slip that is so inserted (Bahr, Axenfeld and Schürenberg).

D. Somewhat analogous to the condition just described is that known as *strabismus fixus*. In this the eye is turned strongly in and cannot be turned outward even by traction with forceps (Berry). Operation has no effect, the eye being evidently fixed in its deviating condition by strong adventitious bands. This condition tends to grow worse in after life and is not always congenital (Fisher, Donaldson, De Vincentiis).

There are certain *peculiarities distinguishing congenital from acquired paralyses*. These are according to Kunn (cited by Wilbrand and Saenger, and Groenouw):

(1) Absence of secondary contracture (page 243). This is a usual but not an invariable sign, for secondary contracture may be absent in acquired and present in congenital paralysis (A. Graefe). Corresponding to this absence of contracture is the fact that there may be no deviation in the primary position, or there may be a deviation contrary to what we should expect. But such an anomalous placing of the eyes in congenital paralysis seems, often at least, to be due to complications which would produce a like result if the paralysis were acquired.

(2) Retention of convergence in spite of the loss of ability to perform lateral movements. This is by no means a sure differential sign.

(3) Absence of secondary deviation in the sound eye. But many cases of congenital paralysis present a well-marked secondary deviation (Duane).

(4) Absence of spontaneous diplopia. This again is by no means the case in all congenital paralyses.

(5) Sudden spasmodic action of the apparently paralyzed muscle.

(6) Absence of any involvement of the iris and ciliary body. This speaks strongly for an orbital origin of the paralysis (structural paralysis). If the paralysis were nuclear we should expect the interior muscles to be at least occasionally affected.

A point laid down by Werthheim (cited by Zweig) is that if ptosis is present, the levator will show a reaction of degeneration in acquired but not in congenital paralysis.

Other alleged differential points, such as the frequent presence of nystagmus or of impaired vision in cases of congenital paralysis, are of no real diagnostic value.

2. **Heredity.**—The effect of heredity is seen in some forms of congenital paralysis, particularly ptosis, which show a distinct tendency to run in families. The same thing is occasionally seen in acquired paralysis,—*e.g.*, in acquired ptosis (Delord) and in a case reported by Beaumont, in which ophthalmoplegia exterior occurred regularly in several members in each of four generations of the same family.

Heredity also plays an important part in inducing the conditions (arteriosclerosis, syphilis, etc.) which frequently lead to paralysis (see *infra*).

3. **Traumatism.**—This is a fairly frequent cause of paralysis. Paralysis of the *external rectus* is most frequent and may be bilateral. It is sometimes evidently orbital in origin, being due to injury of the muscle itself or the orbital section of the abducens nerve; at other times, especially when bilateral, is basal, being due particularly to fractures through the petrous bone. Traumatic paralysis, complete or partial, of the *third nerve* is also often basal, being due to fracture of the base of the skull or penetrating wounds through the orbit or mouth. Isolated paralysis of the *superior* or *inferior rectus* or of the *inferior oblique* is also frequently (although not, as has been asserted in the case of the latter muscles, invariably) the result of traumatism. The injury here usually affects the muscle itself or the nerve-terminals in the orbit (orbital paralysis). The same is true of the not infrequent traumatic *ptosis.*

Traumatic paralysis of the *superior oblique* is rather uncommon. It may be basal or orbital—one variety of the latter being the paralysis sometimes observed after operations upon the frontal sinus and caused by injury to the trochlea.

Traumatic paralyses are due either to *direct injury* of the muscle or nerve, in which case they are found immediately after the injury, or to *compression* by displaced bone, effused blood, callus, traumatic cerebral abscess, or a traumatic aneurism (especially of the internal carotid), in which case they may not develop until some time later. In some cases, but probably not often, traumatic paralysis is *nuclear,* being due, according to Bernheimer, to distention of the ventricles consequent upon the injury and leading to hemorrhage into the nuclei adjoining.

As above noted (page 222), traumatic paralysis may be *congenital.*

Some cases of traumatic paralysis, especially when due to injury of the face, are associated with *enophthalmos.* This, as Purtscher says, may be caused by (*a*) retraction due to scar-tissue; (*b*) loosening of the check-ligaments or the insertions of the obliques; (*c*) displacement of the contents of the orbit; (*d*) paresis of the sympathetic (in this case there will be unilateral miosis); (*e*) trophic changes from injury to the peripheral nerves.

One variety of traumatic paralysis that we meet with pretty often is that *produced by a tenotomy.* We must, in fact, remember that whenever we produce an effective tenotomy we really produce a paresis of the muscle. The action of the check-ligaments and of the adminicula of the tendons prevents this paresis from becoming complete, and when the tendon re-attaches itself it is able to work on the eyeball as before, although not as effectually. Nevertheless, even after re-attachment, if the operation has had any effect at all, we can usually demonstrate restriction of movement such as would be caused by paresis of the muscle, and in many cases also a characteristic diplopia. This is especially seen after tenotomies of the superior and inferior recti. In these for years after the original operation we can in many cases demonstrate diplopia in one quadrant of the field of fixation.

The *fact that a tenotomy has caused a paralysis* may often be inferred from (*a*) the presence of a conjunctival scar or of sub-conjunctival adhesions. If not demonstrable by inspection these can be readily made out by exploration through a trifling and harmless incision in the conjunctiva made under cocaine. (*b*) There may be slight exophthalmus, particularly when the eye is at rest. (*c*) In paralysis of the superior or inferior rectus, due to a tenotomy, the palpebral fissure is wider than normal, since with the retraction of the tendon, its palpebral band also retracts and pulls the lid back. In paralysis of these muscles due to any other cause, the palpebral band is drawn upon less than usual, because the muscle to which it is attached is flaccid, and hence the palpebral fissure is narrower than normal.

4. **Vascular Changes in the Brain.**—Paralysis, both basal and nuclear, may be caused by—

(*a*) *Hyperæmia* and *anæmia* of the brain. These produce often very transient paralyses (Bernheimer).

(*b*) *Embolism.*

(*c*) *Sclerosis of the vessels* due to senile changes, syphilis, lead-poisoning, etc., causes paralysis by pressure or more often by hemorrhage due to their rupture.

(*d*) *Aneurisms* either (1) of the internal carotid or posterior cerebral, or (2) miliary aneurisms of the small vessels. The former act by pressure. Aneurism of the internal carotid causes usually gradual paralysis of the third nerve, combined with paralysis of the fifth; less often paralysis of the fourth and sixth nerves. Miliary aneurisms, which are frequently due to syphilis or to the effect of alcohol, tobacco, or other deteriorating influences, give rise to hemorrhages producing basal or nuclear paralyses.

5. **General Distention of the Brain, or Increase of Intracranial Pressure.**—Abscesses or tumors in the interior of the brain, or other processes (hydrocephalus) which are not so situated as to cause direct pressure on the nerves, yet may induce paralysis by indirect pressure, especially if at the same time there are other conditions (arteriosclerosis, etc.) which favor the development of paralysis. In such a case lumbar puncture may relieve the condition (Brieger cited by Eversbusch).

6. **Inflammation of the Brain and Membranes.**—(*a*) *Encephalitis* of adults and of children (inflammatory cerebral softening, cerebral infantile paralysis) is an occasional cause of paralysis.

(*b*) *Abscess of the brain,* either traumatic or otitic; especially the otitic abscess of the temporal lobe, which may cause paralysis of the third (rarely of the sixth) nerve. It is often associated with other evidences of pressure, especially optic neuritis and a crossed hemiplegia.

(*c*) *Polioencephalitis superior,* a condition of hemorrhagic infiltration of the gray matter about the ventricles, producing destruction first of the association tracts and then of the nuclei, and ending in complete or nearly complete ophthalmoplegia. It occurs particularly in drinkers, but may also be caused by influenza. It is usually but not invariably fatal.

(*d*) *Meningitis,*- syphilitic, tuberculous or cerebrospinal. The nerves at the base may be affected either because they are compressed by the exudate or because they become secondarily inflamed. Cerebrospinal meningitis is especially apt to cause abducens paralysis and ptosis.

7. **Other Diseases of the Brain producing Ocular Paralyses.**—(*a*) *Acute bulbar paralysis* causing often paralysis of the abducens, and not infrequently of the oculo-motor nerve also.

(*b*) *Myasthenia gravis.* In this evidences of paresis of the ocular muscles (especially ptosis) are very frequent and are often the initial symptoms.

(*c*) *Progressive amyotrophic bulbar paralysis* and *pseudobulbar paralysis* very rarely cause ocular paralyses.

8. **Tumors of the brain or skull,** especially glioma, tuberculoma, and

gumma, of the brain; gumma, exostosis and sarcoma in the cranial cavity and orbit; and especially tumors of the hypophysis (acromegaly.) In some cases, according to Bernheimer, these tumors act not only by causing pressure, but also by virtue of the *toxins* which they discharge into the circulation. In cases of basal paralyses due to tumors, the third nerve is particularly apt to be affected. Involvement of other cranial nerves (second, fifth, seventh, and twelfth) is common, and optic neuritis is very frequently although not invariably present.

Tumors of the *pons* readily give rise to abducens paralysis, often combined with paralysis of the facial and with crossed hemiplegia, and not infrequently with involvement of the fifth, eighth, and twelfth nerves.

Rarely, as we have already seen (page 219), paralysis may be due to *metastases of tumors* taking place in the nerves or the muscles themselves.

9. **Spinal Diseases.**—Chief among these are—

(*a*) *Tabes.*—This is a very frequent cause of paralysis of the eye muscles both interior (see Chapter XII) and exterior. According to Uhthoff, one-fifth of all ocular paralyses are tabic and rather more than one-fifth of all tabic patients have ocular paralyses. Partial or complete paralyses of the third nerve are the most common (54 per cent.), next comes paralysis of the abducens (33 per cent.), and of the superior oblique (8 per cent.), while in 5 per cent. all the exterior muscles are affected. In about one-quarter of the cases the optic nerve is simultaneously affected, and in 10 per cent. there is facial paralysis.

Tabic paralyses are apt to occur early in the disease. This is particularly true of ptosis, which is very frequent indeed, and often, in fact, the only evidence of paralysis.* In any case, but especially when occurring early, tabic paralyses are usually incomplete, fluctuating and temporary. Often the paralysis affects only one or two muscles, and these incompletely; then, after lasting for a period varying from a few days to several months, and, after extending perhaps to other muscles in the same eye or its fellow, it disappears spontaneously. Recurrences are frequent either in the muscles originally affected or in others. The Argyll-Robertson pupil is usually found and the knee-jerk is usually absent.

Paralyses which develop later in the course of the disease are apt to be both more complete and more lasting, and some are absolutely permanent. A fair proportion of the cases of progressive, ultimately total ophthalmoplegia are tabic.

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* Fraenkel thinks that in many of these cases of tabic ptosis, especially when very transient, there is no real paralysis, the patient failing to lift the lid simply because, owing to the accompanying anæsthesia, he cannot tell whether it is raised or not. To this condition may be given the name of *pseudoptosis anæsthetica*.

A tabes which has been preceded by syphilis or one which is combined with psychic symptoms is much more likely to cause eye-paralyses than an uncomplicated tabes.

Most tabic paralyses are nuclear, although some are undoubtedly basal and even orbital in origin. Strümpell thinks that the initial, transitory paralyses are especially apt to have a peripheral site.

(*b*) *Combined Lateral and Posterior Sclerosis.*—This not infrequently produces paralysis of the eye muscles.

(*c*) *Multiple Sclerosis.*—Paralysis of the ocular muscles occurs very frequently in this disease (according to Uhthoff in from 17 to 20 per cent. of the cases). Paralysis of the abducens (sometimes bilateral) is most frequent; trochlear paralysis is very rare.

As in tabes, paralysis often occurs early, being sometimes the initial symptom of the disease; and also, as in tabes, the paralysis is apt to be incomplete and transitory. Indeed, a complete and permanent paralysis is very rare.

An associated symptom of considerable diagnostic importance is nystagmus or pseudonystagmus (see Etiology of Nystagmus in Section V, page 278).

The site of the paralysis is usually nuclear, but sometimes peripheral.

(*d*) *Syringomyelia.*—Ocular paralyses, especially paralysis of the abducens, occur in about 11 per cent. of all cases of syringomyelia (Uhthoff). Here also true nystagmus is a frequent, associated symptom (Compare Chapter XV).

(*e*) *Acute Ascending Paralysis.*—This not infrequently causes paralysis, due to change in the nuclei or the peripheral nerves, with, in some cases, degenerative changes in the muscles themselves. Bilateral abducens paralysis is most frequently observed; next unilateral abducens paralysis without or less often with paralysis of the third and fourth nerves. Paralysis of the third or fourth nerves without involvement of the abducens is rare (Uhthoff). (Compare Chapter XV.)

(*f*) *Progressive muscular dystrophy* causes a primary orbital paralysis due to atrophic changes in the muscles themselves.

(*g*) *Other spinal diseases,* very rarely causing ocular paralysis, are lateral sclerosis, Friedreich's ataxia, amyotrophic lateral sclerosis, progressive muscular atrophy, poliomyelitis acuta, and myelitis. Chap. XV.

10. **General Paresis.**—Paralyses of the ocular muscles may form a

prodromal symptom of general paresis or may develop after the psychosis has become well established. They are not very frequent and are usually transitory. In 1 to 2 per cent. of the cases, however, a chronic and progressive, ultimately total ophthalmoplegia develops (Chapter XII).

11. **Hysteria, Neurasthenia, and the Traumatic Neuroses** very rarely indeed produce paralysis of the individual muscles, causing rather paralysis or spasm of the associated parallel movements or of convergence. Apart from hysterical ptosis, which is probably in most cases referable to a spasm of the orbicularis, and which in other cases is probably a pseudo-paralysis due simply to anæsthesia of the lid (cf. Fraenkel, p. 228, note), cases of paralyses of the abducens and of branches of the third nerve have been reported. Most of these are doubtful. In Sachs's case, for example, reported as an hysterical ophthalmoplegia, the paralysis apparently affected rather the associated movements than the muscles themselves. But genuine cases of abducens paralysis due to hysteria have been reported as by Duchenne, Leber, Parinaud, and Suker (See Chapter XVI).

It need scarcely be added that diplopia occurring in hysteria is not, without further examination, to be taken as an evidence of paralysis.

12. **General Diseases** causing paralysis are—

(*a*) *Syphilis.*—This, especially in the tertiary stage, is by far the most frequent cause of paralysis. Apart from the post-syphilitic diseases, like tabes, general paresis, and lesions of the vessels (sclerosis and miliary aneurisms), all of which, as we have seen, so often produce ophthalmoplegias, syphilis acts directly to induce paralysis, by causing periostitis or gummy growth in the orbit; periostitis, meningitis, or gummy infiltrations at the base of the brain; and gummata or degenerative processes in or near the nuclei. Probably at least half of the cases of ocular paralysis are due thus directly to syphilis.

Paralysis of the exterior muscles, due to syphilis, is usually basal. The third nerve, in some or all of its branches, is the one most commonly affected, although any or all of the motor nerves of the eye may be attacked. The paralysis often develops very suddenly, and is frequently associated with or preceded by evidences of involvement of other cranial nerves, especially headache and anæsthesia or neuralgia along the branches of the fifth nerve. There may also be optic neuritis and either scotomata or contraction of the visual field, which latter may be more or less symmetrical, especially if, as often happens, the region of the chiasm is occupied by a syphilitic exudate. The paralysis when once developed is often progressive, but may be stationary or show sudden variations, or even disappear altogether. It is not, however, so apt to be transitory, nor so apt to be limited to single muscles as in the case of tabic paralysis (Fournier).

Congenital syphilis very rarely indeed causes paralysis. Cases, however, have been reported (Lawford, Zappert, and Schmidt-Rimpler cited by Bernheimer, who also reports a case; Cruchandeau).

(*b*) *Diphtheria.*—About 5 or 6 per cent. of all cases of diphtheria are followed (usually in the course of three or four weeks) by a paralysis of accommodation, which is generally bilateral and is generally unaccompanied by paralysis of the sphincter.

Probably some 2.5 per cent. (Goodall, cited by Groenouw) of the cases of diphtheria are followed by paralysis of some of the exterior muscles of the eyeball. Much the most frequent form is paralysis of the abducens, which is often bilateral. This is frequently associated with paralysis of the accommodation. Complete paralysis of the third nerve is very rare (two of Tellais's cases and one of Parkinson's cited by Groenouw), and partial third-nerve paralysis is uncommon.

Cases of trochlear paralysis have been described by Graefe and Krause. Uhthoff and others report a number of cases in which all the exterior muscles of both eyeballs (mostly without involvement of the accommodation) have been attacked.

Other paralyses, especially of the soft palate, and not infrequently of the body-muscles (hemiplegia and paraplegia) may occur at the same time with the paralysis of the ocular muscles.

Paralysis of the exterior muscles of the eyes is especially apt to follow severe cases of diphtheria, while paralysis of accommodation may occur in connection with very light forms of the disease (Groenouw).

The paralysis in some epidemics appears and disappears very suddenly, leaving one muscle to attack another (Pagenstecher, cited by Groenouw). In almost all cases the paralysis ultimately disappears altogether. Mühsam (cited by Groenouw), however reports one case of permanent paralysis of acommodation; and Morvat and I have reported cases of permanent paralysis of the exterior muscles.

In at least three of the reported cases (Groenouw, Tellais), death ensued soon after the development of the paralysis.

The lesion causing the paralysis has been variously ascribed to capillary hæmorrhages in the nerve-nuclei, disease of the blood-vessels supplying the nuclei or nerves, basal or orbital neuritis, and inflammatory changes in the muscles themselves (Groenouw).

(*c*) *Influenza* often causes a paralysis of accommodation, and paralyses of the exterior muscles are not very infrequent. The abducens is especially apt to be affected, although paralysis of some or all of the muscles supplied by the third nerve is also observed. Isolated trochlear paralysis is very rare. In some instances there is paralysis, complete or partial, of all the muscles moving the eyeball.

The paralysis of the accommodation is usually bilateral, the paralysis of the exterior muscles unilateral, although bilateral paralysis of the abducens (Bernheimer), the trochlear (Pflüger), and the third nerve have been reported.

In these paralyses recovery is the rule. In some cases, however, especially when the exterior muscles are involved, the paralysis remains indefinitely.

The lesions causing the paralysis are supposed to be neuritis, nuclear or basal hemorrhages, circumventricular hemorrhages with the picture of a polioencephalitis superior (page 227), and a true encephalitis. Influenza may also cause paralysis by inducing an orbital cellulitis or an inflammation of Tenon's capsule.

(*d*) *Tuberculosis* may cause paralysis by inciting a tuberculous meningitis (page 227). Otherwise, paralysis is very rarely indeed the result of tuberculosis, although now and then it may be induced by tuberculous foci situated at the foramina of exit of the nerves, or at the base of the skull or in the brain itself. The paralysis is usually basal or foraminal, very rarely nuclear.

(*e*) *Rheumatism.*—Acute articular rheumatism occasionally produces paralysis, mostly as a result of the metastatic processes (meningitis, orbital cellulitis, tenonitis) to which it gives rise.

Much more frequent are certain paralyses to which the name *rheumatic* is usually applied; occurring generally *as a result of exposure to cold,* and especially in those who are the subjects of chronic rheumatism. The paralysis usually develops suddenly and is often associated with various rheumatic manifestations, with neuralgic pains along the course of the fifth nerve, and sometimes with paralysis of the facial. The abducens is most commonly affected, but paralyses of the other nerves, complete or partial, are not infrequent.

Such paralysis is usually quite amenable to treatment with salicylates and by diaphoresis.

(*f*) *Herpes zoster ophthalmicus,* especially the forms that are associated with fever or are due to toxic conditions, often cause paralysis. This usually affects isolated muscles supplied by the third nerve, although sometimes all the branches of the latter are attacked. The fourth and sixth nerves are rarely affected.

(*g*) *Typhoid Fever.*—Paralysis of any of the motor nerves of the eye may develop in typhoid fever, usually after the second week but sometimes earlier. They are ascribed to complicating affections of the brain or meninges or to a peripheral neuritis (Groenouw), but may perhaps also be due to hemorrhage. They are sometimes transient but may be permanent.

(*h*) *Cerebrospinal meningitis* (page 227).

(*i*) *Malaria* very rarely causes an oculo-motor or an abducens paralysis, which sometimes recurs periodically with the fever and which disappears under the use of quinine.

(*j*) *Gerlier's disease* (a peculiar disease endemic in Switzerland, marked by recurrent attacks of vertigo and paresis of the extremities) often produces ptosis and paralysis of the abducens or of the individual branches of the third nerve. A somewhat similar, but often fatal, affection is *kubi-sagari,* endemic in Japan. In this, too, ptosis and other ocular paralyses are frequent.

(*k*) *Beriberi* often produces paralyses which are usually transient.

(*l*) *Other infectious diseases* occasionally producing paralysis are pneumonia (Alt, Aldrich); tetanus and cephalic tetanus; Weil's disease (Groenouw); mumps; whooping-cough (Gowring, Batten); erysipelas and pyæmic states. These probably act mainly by their secondary effects (meningitis, orbital cellulitis, hemorrhages due to convulsions).

13. **General conditions not essentially infectious** are—

(*a*) *Diabetes.*—In some dozen recorded cases, paralysis of the eye muscles has been caused by diabetes mellitus, and in one or two instances by diabetes insipidus. In the latter case there was probably a lesion about the fourth ventricle, causing both the polyuria and the paralysis.

(*b*) *Renal Disease.*—A few cases of paralysis due to this cause have been reported by Dunn, and by other authorities (Knies, Bernheimer). They are in all probability due to hemorrhage. They appear usually to indicate a fatal issue.

(*c*) *Exophthalmic goitre* occasionally causes paralysis of the abducens and less often of the exterior muscles supplied by the third nerve.

(*d*) *Acromegaly.*—Apart from the rather frequent cases of paralysis produced by the tumors of the hypophysis in acromegaly (page 228), paralysis is sometimes apparently due to some influence that this disease exerts upon the general condition (Bernheimer). The abducens and trochlear nerves are rarely, if ever, affected.

(*e*) *Auto-intoxications* from the digestive tract very rarely cause paralysis of the exterior muscles.

(*f*) *Profuse Hemorrhage.*—Two cases of bilateral abducens paralysis due to this cause are recorded by Neuburger.

14. **Poisons.**—There are a number of poisons which produce paralysis of the interior muscles of the eye (sphincter and ciliary muscle). For these, called cycloplegics, reference must be had to Chapter VI. Paralysis of the exterior muscles, on the other hand, is rarely caused by poisons.

(*a*) *Alcohol.*—Chronic alcoholism in a few cases (about 0.5 per cent., according to Uhthoff) produces paralysis. Bilateral paralysis (usually incomplete) of the abducens is the most common form. This seems to be due to a nuclear lesion.

Alcoholism also appears to play an important part in the genesis of polioencephalitis superior, which, as already stated (page 227) is associated with a progressive, ultimately total ophthalmoplegia.

(*b*) *Sulphuric acid* may in very rare instances cause paralysis by producing polioencephalitis superior.

(*c*) *Gelsemium* often causes ptosis and diplopia.

(*d*) *Other poisons* occasionally producing paralysis of the exterior muscles are tobacco (cases not certain, although Wilkinson's case seems authentic); atropine (Baas); snake-venom; lead; carbon monoxide (illuminating gas); fungi; and decayed meat, fish, oysters, etc. (ptomaine-poisoning). In ptomaine-poisoning ptosis is comparatively frequent.

15. **Diseases of the nose, and especially of its accessory sinuses,** produce paralysis, mainly by mechanical pressure, but sometimes apparently because of resorption of pent-up purulent matter. The same is apparently true of some cases of *otitis,* which produce paralysis, not by the pressure due to an otitic abscess (page 227), but by the resorption of infective material (Bernheimer).

Diagnosis of Cause.—1. **Most Frequent Causes.**—Of the long list of causes above given, the ones most frequently encountered are syphilis, tabes, multiple sclerosis, exposure to cold (rheumatic paralyses), traumatism, meningitis, tumors and abscess of the brain, arteriosclerosis and aneurisms (producing pressure or hemorrhage). Less frequent are general paresis, diphtheria, influenza, alcoholism (especially as inducing polio-encephalitis).

A *suddenly developing* paralysis is usually due to exposure to cold, traumatism, hemorrhage, embolism, or thrombosis. In some cases of diphtheria the paralysis also appears and disappears suddenly.

Incomplete and temporary, more or less fluctuating, paralyses are the special characteristic of tabes (particularly in the early stages) general paresis, and multiple sclerosis. Post-diphtherial and rheumatic paralyses are also usually temporary, and traumatic paralyses often so.

2. **Abducens Paralysis** is frequently caused by syphilis, tabes, and tuberculous meningitis, although these conditions still more often produce oculo-motor paralysis. On the other hand, traumatism, exposure to cold, cerebrospinal meningitis, multiple sclerosis, syringomyelia, acute ascending paralysis, acute bulbar paralysis, pons lesions, diphtheria, influenza, and alcoholism much more often cause paralysis of the abducens than of the exterior muscles supplied by the third nerve. Congenital paralysis also often affects the abducens. In some of the conditions here noted, especially acute ascending paralysis, diphtheria, and alcoholism, *both abducens nerves* are often affected simultaneously.

3. More or less **complete paralysis of the third nerve** is very often due to syphilis. Other conditions particularly apt to cause this form of paralysis are tumors and abscesses of the temporal lobe, tumors at the base of the brain (especially the hypophysis tumors of acromegaly), and aneurism of the internal carotid. Complete paralysis of the third nerve is not common in tabes, and bilateral complete third-nerve paralysis is hardly ever tabic (Uhthoff).

4. **Paralysis of isolated branches of the third nerve** is found especially in tabes and herpes zoster, but is also often due to syphilis, and may be caused by traumatism or be congenital.

5. One form of isolated paralysis of the third nerve,—namely, **ptosis,**—is of special diagnostic importance.

Ptosis *without paralysis of any of the muscles moving the eyeball* occurs most often under the following conditions:

(*a*) As a congenital state. Here usually bilateral, often incomplete, and always permanent.

(*b*) As a condition developing without assignable cause in old age. Always bilateral, gradual in development, and unassociated with any other evidence of nerve trouble.

(*c*) In tabes. Frequent, especially in initial stage of tabes. Usually incomplete and transitory. May be a pseudoptosis (page 228 note).

(*d*) In multiple sclerosis. Rare.

(*e*) As an initial symptom of chronic progressive ophthalmoplegia.

(*f*) In syphilis. Rare.

(*g*) In cerebral softening (embolic or traumatic). Quite frequent.

(*h*) In cerebral abscess, particularly otitic abscess of the temporal lobe on the same side as the ptosis. Here often associated with paresis of the opposite side of the face and arm, sometimes of the leg. The same combination may occur in tumors of the brain.

(*i*) In cerebrospinal meningitis. Not uncommon.

(*j*) In cerebral hemorrhage. Occasionally.

(*k*) Traumatism affecting the lid or levator directly, or occasionally traumatism producing intracranial lesion (even fracture of the base).

(*l*) As evidence of a cortical lesion. (Usually in or near the angular gyrus on the opposite side to the ptosis). Rare.

(*m*) In hysteria. Usually bilateral.

(*n*) In ptomaine-poisoning. Associated with mydriasis and paralysis of accommodation.

(*o*) A slight degree of ptosis is produced by paralysis of the superior rectus, provided this is not due to division of the tendon, a lesion which causes retraction of the lid (page 226).

Ptosis *with more or less involvement of the extra-ocular muscles supplied by the third nerve* is found often in tabes, syphilis, cerebral softening, cerebral abscess, tumors in various parts of the brain, meningitis (especially tuberculosis, Gerlier's disease, polioencephalitis superior, and as a result of traumatism.

A *bilateral incomplete ptosis,* which, slight in the morning, increases markedly in the evening, is characteristic of myasthenia gravis.

Ptosis *associated with abducens paralysis* (with or less frequently without involvement of the extra-ocular muscles supplied by the third nerve) is very frequent in tabes.

In using ptosis as a diagnostic sign we must be careful to exclude those conditions in which a drooping of the lid obtains but *is not due to third-nerve paralysis.* Such conditions are—

(*a*) *Sympathetic Ptosis.*—In this the palpebral fissure is moderately contracted, but as much by raising of the lower lid as by drooping of the other. Repeated instillations of cocaine will not widen the palpebral fissure as they will in a third-nerve ptosis. There is usually miosis, and the pupil is undilatable by cocaine. There may be hyperidrosis or anidrosis with redness or pallor of the face on the side of the lesion. This kind of ptosis is quite frequent in tabes.

(*b*) *Pseudoptosis from spasm of the orbicularis* (spastic ptosis) occurs in irritative conditions of the eye, and is the usual form of ptosis in hysteria. Perhaps also the ptosis observed in pons lesions is due to this cause (Schoeler). In this form, as in sympathetic ptosis, both lids, the lower as well as the upper, take part in producing contraction of the palpebral fissure.

(*c*) Drooping of the lid from mere *excess of weight or mechanical causes* as in tarsitis syphilitica, trachoma, marked œdema, etc.

(*d*) The *pseudoptosis anæsthetica* (Fraenkel, page 229, note).

(*e*) The slight *drooping of the lid produced by an advancement* of the superior rectus. This operation, by carrying the tendon forward, draws forward also the superior palpebral band and with it the upper lid, which is thus depressed one or two millimetres. Similarly, advancement of the inferior rectus causes a quite perceptible elevation of the lower lid.

6. **Paralysis of the exterior muscles supplied by the third nerve** without involvement of the fourth or sixth nerves is rare in acute ascending paralysis, syringomyelia, acute bulbar paralysis, diphtheria, influenza, and alcoholism.

7. **Isolated trochlear paralysis** is most often due to syphilis, tabes, meningitis, sometimes to traumatism (especially injuries about the trochlea). It is seldom the result of diphtheria, influenza, or pons lesions, and is very rarely caused by multiple sclerosis.

8. **Simultaneous paralysis of the third and sixth nerves** on the same side ocurs in fractures of the base, especially one which traverses the petrous bone and splits off the clinoid process.

9. **Total ophthalmoplegia,**—*i.e,* the condition in which all three of the motor nerves of the eyes are paralyzed,—is most frequently due to syphilis, tabes (especially the later stages), orbital disease, and traumatism. It is very rare in general paresis, diphtheria, and influenza.

10. **Ophthalmoplegia exterior** occurs often in syphilis, also in tabes, and various basal and nuclear diseases. It is usually bilateral in polioencephalitis superior and the chronic progressive ophthalmoplegia of Graefe (page 242).

Diagnosis of Site.—The following distinctive signs often obtain in cases of **Orbital paralysis.**

1. The paralysis is usually unilateral.

2. It frequently affects a single muscle.

3. The interior muscles are not usually affected, except in the case of a paralytic mydriasis produced by a blow upon the eye itself, or a sympathetic miosis produced by injury to the orbit.

4. In traumatic cases, particularly, there may be enophthalmos often associated with a sympathetic miosis (page 226).

5. In tumors or inflammatory processes in the orbit there is often exophthalmos or a lateral displacement of the eye.

6. Other evidences of orbital lesion are often present, such as scars in the lids or the margin of the orbit, depressed bone, orbital swelling

appreciable by palpation, deep pain elicited by pressure made into the orbit, chemosis, evidences of disease of the accessory sinuses.

It must, however, be remembered that the presence of these *evidences of orbital disease is not proof positive that the paralysis itself is orbital in site.* Thus:

(*a*) Exophthalmos may indicate a paralysis due to aneurism of the internal carotid or a thrombosis of the cavernous sinus.

(*b*) Paralysis may occur with shot or stab wounds of the orbit because the skull has been penetrated and the nerves injured at the base.

(*c*) Paralysis occurring in an orbital cellulitis or orbital tumors may be caused by an extension of the morbid process backward into the cranial cavity and involvement of the nerves at the base.

(*d*) Paralysis associated with marked evidences of orbital disease may, especially if syphilitic, be due to an intracranial process which after affecting the nerves at the base extends into the orbit. This is particularly apt to be the case if the process started by involving isolated muscles, especially those supplied by the third nerve.

A total oculo-motor paralysis with completely intact fourth and sixth nerves is rarely, if ever, orbital (Wilbrand and Saenger).

On the other hand, paralysis of all three motor nerves of the eye without involvement of other cranial nerves except the first branch of the fifth is almost certainly orbital or foraminal in origin.

The following points are useful in diagnosticating a **Basal paralysis.**

1. Paralysis of the other cranial nerves are often present (see next section on Affections of Other Nerves).

2. In traumatic paralysis there are often evidences of fracture of the base (discharge of cerebrospinal fluid, bleeding from the ears). There may also be blood in the fluid obtained by lumbar puncture.

3. In paralysis due to meningitis there are retraction of the head, fever, delirium, and severe headache, and leucocytes are present in the fluid obtained by lumbar puncture.

4. In paralysis due to otitis there may be found, besides the evidences of ear disease with the accompanying deafness and vertigo, the signs of an abscess in the temporal lobe (optic neuritis, crossed facial paralysis and hemiplegia, aphasia, word-deafness, mind-blindness, homonymous hemianopsia). Similar signs would be found in temporosphenoidal tumors.

5. Complete or nearly complete third-nerve paralysis, especially if bilateral, is usually basal or foraminal.

6. Recurrent ophthalmoplegia (ophthalmoplegic migraine—see Chap. XVII) is usually due to basal lesions.

7. It was formerly thought that an ophthalmoplegia exterior and isolated paralyses of muscles supplied by the third nerve must be nuclear, but in a number of cases these conditions have been shown to be due to basal lesions (Uhthoff, Bernheimer, Salomonsohn, Hochwart).

Many of the cases of what were thought to be **nuclear paralysis** are really fascicular, and not a few of the rest are really basal. If, as is generally the case in tabes and multiple sclerosis, the paralysis is evidently due to central causes, it is probably fascicular if more than one nerve is involved. This rule, however, is not without its exceptions, as several instances of multiple nuclear paralyses have been noted (Bernheimer).

The diagnosis of paralysis due to a lesion of the *pons* is based on the presence of the following:

1. Paralysis of one (sometimes both) abducens nerves, or more frequently a paralysis of conjugate movement to the right or left (pages 221 and 270).
2. Optic neuritis (in 25 per cent. of the cases).
3. Associated paralysis of the fifth, seventh (very frequent), eighth, or twelfth nerves.
4. Ptosis (in some cases).
5. Paralysis or paresis on the opposite side of the body. Very common. Sometimes also there are convulsive movements or general convulsions and sensory disturbances.

Affections of Other Nerves as Determining the Diagnosis.

1. **Olfactory Nerve.**—Paralysis of this, causing anosmia, occurs in paralyses associated with nasal disease and in tabes.

2. **Optic Nerve.**—An *optic neuritis* associated with paralysis of the eye muscles suggests, in most cases, the probability that the paralysis is due to tumor or abscess of the brain or to syphilitic or tuberculous meningitis. Less often it occurs in paralysis due to multiple sclerosis or to morbid processes in the orbit, implicating the optic nerve.

Total *optic-nerve atrophy* occurs in at least 25 per cent. of paralyses due to tabes and is also common in paralysis resulting from orbital inflammation or tumors (then usually associated with exophthalmos), traumatism (especially fractures at the apex of the orbit), and periostitis of the sphenoidal fissure. A usually partial (temporal), but sometimes total optic atrophy often accompanies the motor paralyses produced by multiple sclerosis.

Homonymous hemianopsia occurs in paralysis due to basal lesions affecting the optic tract, brain tumors, and otitic abscesses of the temporo-sphenoidal lobe.

Bitemporal hemianopsia may occur in paralyses due to chiasm lesion (especially acromegaly).

Concentric contraction of the visual field occurs in tabic paralyses and irregular contraction or a central scotoma often in multiple sclerosis.

3. **Trigeminus.**—*Paralysis of all branches of the fifth* combined with paralysis of the third (less often of the fourth or sixth) nerves occur especially in syphilis, in tabes, in tumors of the base, and in aneurism of the internal carotid.

Paralysis of the *first branch of the fifth* (with perhaps keratitis neuroparalytica) combined with paralysis of the third and with optic-nerve atrophy indicates disease (syphilitic periostitis) in the sphenoidal fissure.

Paralysis of the fifth combined *with paralysis of the abducens* occurs especially in pons lesions and in syringomyelia.

Neuralgia of the fifth occurs often in syphilitic and rheumatic paralysis.

4. **Facial.**—Paralysis of the facial occurs conjointly with paralysis of the eye muscles as a result of

(*a*) *Tabes* (in about 10 per cent. of tabic paralyses).

(*b*) *Otitis* and particularly otitic abscess of the temporo-sphenoidal lobe, in which case the most usual combination is a paralysis (generally partial) of the third nerve on the side of the abscess and paralysis of the opposite facial. Similar conditions may be produced by a *tumor* situated in the frontal or temporal lobes.

(*c*) *Exposure to cold.* Here the most frequent combination is paralysis of facial and abducens.

(*d*) *Traumatism,* especially fracture through the petrous bone implicating the facial and the abducens or sometimes the facial and the third nerve together.

(*e*) *Pons lesions.* Paralysis of the facial (usually the upper facial being exempt) occurs in about two-thirds of the cases of pons disease, and is then very frequently combined with paralysis of the abducens.

(*f*) *Acute ascending paralysis* (not infrequent) and occasionally also in other spinal diseases (myelitis, etc.). The most usual combination is paralysis of the facial and abducens.

5. **Auditory.**—Paralysis of the eighth nerve (nerve deafness, may occur combined with eye paralyses in otitis, fracture of the petrous bone, syphilis, tumors of the auditory nerve, and pons lesions (in the latter case in conjunction with abducens paralysis).

6. **Hypoglossal.**—This is very often involved in conjunction with the

abducens of the same or the opposite side in pons lesions; sometimes also in lesions situated in the posterior cranial fossa near the middle line.

7. **Hemiplegia.**—Paralysis of the *third nerve with complete hemiplegia of the opposite side* of the body indicates usually either a lesion in the peduncle, or an otitic abscess in the temporo-sphenoidal lobe (in the latter case often also combined with paralysis of the opposite facial), or a tumor in the frontal or temporal lobe.

Paralysis of the *sixth nerve with hemiplegia of the opposite side* of the body indicates a lesion of the pons situated on the same side as the paralyzed abducens.

Course and Prognosis of Paralyses.—As already stated, some paralyses—especially those due to direct effects of traumatism and to hemorrhage, embolism, thrombosis, and exposure to cold—*begin suddenly.* This is also the case with many diphtherial and some syphilitic and tabic paralyses.

In other cases the paralysis *develops slowly*—sometimes very slowly indeed, taking many years before it reaches its height.

The *duration* of a paralysis varies from a few hours to a lifetime. A similar variation is seen in the *completeness* of the paralysis, which may range from a slight restriction of movement of a single muscle to a total ophthalmoplegia producing absolute immobility of the eyeball.

Transitory paralyses are the rule in tabes and multiple sclerosis. The paralysis is also usually temporary when due to diphtheria, exposure to cold, beriberi, and ptomaine-poisoning; and often so when caused by general paresis and influenza. Traumatic paralysis is frequently temporary if due to the pressure of effused blood, which can undergo absorption, to division of a tendon which subsequently becomes re-attached, or to some similar remediable condition.

Paralyses which occur *early in the course of a central nervous disease*—particularly tabes, multiple sclerosis, and syringomyelia—are apt to be less severe and less permanent than those developing in the later stages. The same is probably true, although perhaps not so markedly, of syphilitic paralyses. In syphilis, however, the paralyses usually occur late in the disease, and in any case, although they vary greatly in intensity and duration, are generally more lasting, more complete, and more likely to involve a number of muscles than are the paralyses found in tabes and multiple sclerosis.

Congenital paralyses usually *remain unchanged* through life. An exception seems to be strabismus fixus (page 224), which, even when congenital in origin, may be aggravated by consecutive changes in adult life.

Traumatic paralyses, too, are apt to remain stationary, especially if complete, and the same is true of other paralyses when a cause that has ceased to act has produced complete destruction of a nerve or muscle.

In some cases a paralysis shows a *sudden and great increase.* This may occur in incomplete traumatic paralyses very soon after the injury, and is then referable to a sudden giving way of the adhesions or of a severed but re-attached tendon. But it may also occur, and without apparent cause, in paralyses whether traumatic or not, which have remained stationary for a long time.

In some cases an *apparent sudden increase* in a paralysis is produced by the fact that the patient *fixes with the paretic eye,* allowing the sound eye to deviate. This, without really changing the muscular conditions, causes a great increase in the deviation (page 215).

If, as sometimes happens (page 217), fixation is performed with each eye alternately, the deviation will show a sudden great increase when the paretic eye is used, and it will seem, as the patient shifts from one eye to the other, as if the paralysis was undergoing sudden and great changes in amount.

In other cases still, a paralysis tends to advance gradually and either continuously or discontinuously (*progressive paralysis*). Cases of this sort in which ultimately nearly all the eye muscles are involved, occur in syphilis and tabes.

A peculiar form of progressive paralysis is the rare *chronic progressive ophthalmoplegia* of Graefe. This is an extremely chronic affection, which often begins in childhood and affects very gradually one muscle after another until after many years the picture of a more or less complete ophthalmoplegia exterior is produced. It may be arrested in any stage of its course. In any case the interior muscles are exempt, and, aside from the ophthalmoplegia, no sign of other disease of the nervous system subsequently develops. The affection is always bilateral.

A paralysis, however caused, may *disappear suddenly.* Much more frequently the *recovery is gradual,* and often enough it is incomplete, a more or less marked paresis—sometimes only a very slight insufficiency—of one or more muscles remaining indefinitely.

Even when the recovery is complete, recurrences may take place. Such recurrences may affect the muscle originally involved, or some other muscle in the same eye, or one or more muscles in the fellow-eye. They are observed especially in tabes and in syphilis, but may also occur in paralysis resulting from exposure to cold and those caused by tumors and aneurisms, syringomyelia, chronic meningitis, multiple neuritis, chronic progressive ophthalmoplegia, and other causes.

Paralyses which disappear and *recur at short, regular intervals* may occasionally develop in connection with malaria and perhaps in other conditions. Very peculiar are the cases described by Salzmann (cited by Wilbrand and Saenger), and by Axenfeld and Schürenberg, in which, with an oculo-motor paralysis, there was rhythmical return of power—indeed an actual spasm—in the levator and in the interior muscles every minute or so. Such conditions are attributed to periodical changes in the blood-supply of the oculo-motor nucleus (Fuchs), or, with more plausibility, to the intermittent pressure of a self-emptying angioma on the trunk of the nerve at the base of the brain (Axenfeld and Schürenberg).

A peculiar variety of recurrent paralysis usually affecting the third, but sometimes the fourth or sixth nerves, is the so-called *ophthalmoplegic migraine* (Chapter XVII). Another affection characterized by intermittent recurrent paralyses is *Gerlier's disease* (Chapter X).

In acquired paralysis, particularly when it is complete and when it has lasted a long time, a consecutive **contracture of the antagonist** to the paralyzed muscle commonly develops. Thus, in paralysis of the externus, the internus becomes contractured, causing a strong deviation of the eye inward. In paralysis of the internus, contracture takes place in the externus; in paralysis of the superior rectus the contracture affects sometimes the inferior rectus, sometimes the superior oblique; and in paralysis of the inferior rectus, it affects either the superior rectus or the inferior oblique.

The effect of the contracture is to *extend the deviation and render it comitant.* That is, a deviation which is originally confined to one portion of the field of fixation becomes spread over all portions. Thus, in paralysis of the right abducens, there is originally a marked inward deviation which increases progressively as the eyes are carried to the right, and diminishes to zero as the eyes are carried to the left. Then a consecutive contracture of the right internus takes place, and now the right eye being pulled strongly inward by this muscle, shows a marked inward deviation in the primary position and also when turned to the left. Ultimately such a case cannot be differentiated from a comitant strabismus convergens of the right eye.

So, too, in a paralysis of the right superior rectus the right eye at first simply drops below the left and does so only when the patient looks up, while when he looks down the two eyes are on a level. Afterwards a contracture of the inferior rectus or the superior oblique takes place, and then the right eye is forcibly carried down, so that it is below the left, not only when the eyes are directed up, but also when they are in the primary position and when they are directed down. The condition is thus transformed from a non-comitant deviation into a more or less comitant vertical squint (strabismus deorsumvergens). Similarly a paralysis of the superior oblique or the inferior rectus may through contracture of the opposing elevators produce a comitant strabismus sursumvergens.

While contracture of the opponent is regularly present in old acquired paralyses, it is as regularly *absent in congenital paralysis.* Both rules, however, have their exceptions (A. Graefe).

In some cases the *paralysis itself may disappear and the contracture persist,* leaving a pronounced spastic deviation (A. Graefe).

More or less variable spasm may occur in the opponents of the paralyzed muscle. Thus, in paralysis of the inferior rectus, a condition which is associated mainly with diplopia in looking down, a transient spasm of the superior rectus or inferior oblique may occur, causing sudden changes in the degree of deviation and producing diplopia in looking up, which diplopia will vary in amount from day to day. In my belief such spasm is the usual forerunner of a permanent contracture and is indeed the cause of it.

Treatment of Paralysis.—In treating a paralysis the first and foremost indication is to *remove its cause.* Accordingly, in paralyses due to syphilis and the parasyphilitic diseases (tabes, general paresis) we use large doses of the *iodides* and *mercury.* In syphilis, where the paralyses often occur late in the disease and indicate a deep-seated lesion, which may be the immediate precursor of even more serious changes in the nervous system, treatment should be pushed very vigorously and kept up for many months. The earlier such thorough-going treatment is initiated, the greater the prospect both of relieving the paralysis itself and of preventing the development of other serious manifestations later on.

In paralyses due to exposure to cold, treatment with *diaphoretics* and *salicylates* is indicated and is usually successful.

In paralyses resulting from diphtheria, influenza, hysteria, and neurasthenia, *tonic treatment* should be employed. Use of the eyes for near work should be restricted or forbidden for a time, at least, so as to give the central neurons a chance to regain their full activity.

Strychnine has been recommended in paralysis, but seems of doubtful value except as a general tonic.

Electricity has been used with success by Benedikt (cited by Beard and Rockwell), by Roy, and others.

Passive traction (made with forceps under cocaine), *exercises* in moving the eye (as in following a moving object), and exercises in overcoming diplopia, either with or without prisms, may be of use in partial paralyses and particularly as an adjuvant when the paralysis shows any tendency to recover. This treatment is much more effective in peripheral (and especially traumatic) paralyses than in those of central origin.

Finally, if the paralysis has remained stationary for months, and there is no apparent prospect of a change taking place in it, an *operation* may be done. This consists in

(*a*) Advancement of the paretic muscle itself. Done especially in partial and in traumatic paralyses.

(*b*) Advancement of another muscle in the same eye, especially of the superior rectus if the inferior oblique is paralyzed.

(*c*) Tenotomy of the direct antagonist in the same eye,—*e.g.*, of the right internus when the right externus is paralyzed. Should be done only in case there is well-marked contracture of the direct antagonist.

(*d*) Tenotomy of the associate muscle (page 194) in the fellow-eye, thus of the left internus, externus, inferior rectus, superior rectus, inferior oblique, and superior oblique, respectively, in the case of paralysis of the right externus, internus, superior oblique, inferior oblique, superior rectus, and inferior rectus. This operation affords very good results.

In many cases a combination of several of these operations may be required.

To obviate the diplopia due to the paralysis many patients find it advantageous to shut out the sight of one eye by putting a *patch* or a *ground glass* before it. Others find these devices intolerable.

Conditions Allied to Paralysis.—**Dyskinesis** is the condition in which the muscles are not paretic and are, in fact, capable of contracting to the full normal extent, yet act sluggishly or with pain and difficulty. It may occur after any exhausting illness, as an evidence of neurasthenia or hysteria, or as one of the symptoms of general muscular enfeeblement from any cause. In some cases it seems to occur without obvious cause and as an independent affection in otherwise healthy people.

A peculiar **ataxia of the eye-muscles** sometimes, although very rarely, occurs in tabes (Uhthoff). If the atactic movements are associated (comitant) so that one eye acts precisely like the other, there will be no diplopia. In this case the condition is an ataxia rather of the ocular movements than of the muscles themselves, and may be regarded as a kind of irregular nystagmus. When the atactic movements differ in the two eyes, so that the eyes make dissociated movements, a peculiarly irregular and changeable diplopia will ensue.

Section III.

SPASM OF THE INDIVIDUAL OCULAR MUSCLES.

Symptoms.—Normally, when the eyes are carried in a given direction, they move equally far and equally fast, because the two associate muscles (page 194) chiefly concerned in that special movement work precisely alike. If one of these muscles is underacting, the eye to which it is attached lags more and more behind the other in performing this special movement, and we have in consequence an increasing deviation, an increasing diplopia, and the other symptoms of paralysis already noted. Quite the same thing will happen if one of the associate muscles is overacting. In that case the affected eye will shoot further and further ahead of the other, and we will have again a deviation and a diplopia, increasing as the eyes are carried in the given direction.

Accordingly, the evidences of a spasm or overaction are as follows:

1. *Excessive Movement.*—The affected eye shoots beyond the other when the eyes are carried in a certain direction, and its total range of excursion in that direction is greater than that of the fellow-eye.

2. The *deviation* thus produced, which can be made out by the screen and other tests, is greater if the sound eye is used for fixation than if the affected eye fixes,—*i.e.*, the *primary is greater than the secondary* deviation. (*Cf.* remarks, page 198, 212).

3. *Diplopia,* increasing as the eyes are carried in the direction of the field of action of the overacting muscles. This diplopia is precisely the same in character and in behavior as that which would be produced by a paralysis of the associate muscles in the fellow-eye,—*i.e.*, the conditions will be as shown in the following table.

SPASM OF RIGHT	CORRESPONDS TO PARALYSIS OF LEFT	IN EITHER CASE			
		IF L. EYE FIXES R. DEVIATES GREATLY	IF R. EYE FIXES L. DEVIATES	DIPLOPIA IS	DEVIATION AND DIPLOPIA INCREASE AS EYES ARE CARRIED
External rectus.	Internal rectus.	Out.	Out.	Crossed.	To R.
Internal rectus.	External rectus.	In.	In.	Homonymous.	To L.
Superior rectus.	Inferior oblique.	Up.	Down.	Right.*	Up and to R.
Inferior oblique.	Superior rectus.	Up.	Down.	Right.*	Up and to L.
Inferior rectus.	Superior oblique.	Down.	Up.	Left.†	Down and to R.
Superior oblique.	Inferior rectus.	Down.	Up.	Left.†	Down and to L.

Note.—The conditions obtaining in spasm of the left eye (paralysis of the right) may be deduced by substituting right for left and *vice versa*.

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* *I.e.*, vertical diplopia with the image of the right eye below.

† *I.e.*, vertical diplopia with the image of the left eye below.

4. *False Projection.*—If the projection test is made in the manner indicated on page 210 by having the patient close the good eye and try to put his finger on an object situated within the field of action of the affected muscle it will generally be found that in a case of spasm the patient undershoots his mark, just as in paralysis he overshoots it. Thus, a patient with a spasm of the right abducens who is trying to touch an object on his right, will put his finger to the left of it. The reason for this is that the patient underestimates the distance that the eye travels, since he is aware only of the effort that he is putting forth in order to look at the object, and with an overacting muscle this effort is less than normal.

5. An *apparent movement* of objects looked at may be produced by the false projection, and this movement together with the diplopia may cause *vertigo.*

As will be seen from the above, the appearances presented by a spasm of a muscle in one eye closely simulate those of a paralysis of the associate muscle in the other eye. Indeed, unless the objective signs of a paralysis, such as marked restriction of movement, are present, the diagnosis may be difficult. It can be facilitated by the following considerations:

Points in Common.

One eye moves faster and further than the other when both are carried in some one particular direction; and both this discrepancy between the position of the eyes and also the diplopia, false projection, and vertigo become more and more pronounced, the further the eyes are carried in that direction.

SPASM.	PARALYSIS.
Measured by the perimeter or tropometer, the absolute excursion of the faster moving eye in the given direction is greater than normal; that of the other eye is normal.	Measured by the perimeter or tropometer, the absolute excursion of the faster moving eye is normal in all directions; that of the other eye is subnormal.
The total excursion of the faster moving eye in the given direction and in the direction opposite is greater than normal,—*i.e.*, the field of fixation is excessively large in one of its diameters.	The total excursion of the faster moving eye is normal, that of the other eye is subnormal,—*i.e.*, the field of fixation of the latter is contracted in one of its diameters.
Fixation is usually performed by the slower moving eye.	Fixation usually performed by the faster moving eye.
The amount of deviation may show great and sudden changes from time to time.	The amount of deviation rarely shows these sudden changes.
If false projection is present, it occurs when the faster moving eye is used for fixation (the other being closed), and the patient undershoots the mark he is trying to touch.	False projection occurs when the slower moving eye is used for fixation, and the patient overshoots the mark he is trying to touch.

Etiology of Spasm.—Overaction of an ocular muscle may occur either because the muscle itself is excessively strong and overdeveloped (*structural overaction*), or because the tendon is inserted so close to the cornea that it exerts an undue leverage on the eye (*insertional overaction*). A marked example of insertional overaction is that produced by the operation of advancement.

True spasm is that form of overaction produced by excessive innervation. It may be either primary or secondary.

Primary spasm of the ocular muscles is rare. A spastic, sometimes painful, contraction of one or more muscles occurs at times in *meningitis and other irritative affections* of the brain. It has also been observed as a result of *reflex irritation* due to decayed teeth (Eversbusch), or to middle-ear disease. Lucae found that artificial heightening of the labyrinthine pressure produces a divergent squint which he ascribes to spasm of the abducens. Tonic spasm also occurs in tetanus. Mackenzie says that in this disease sometimes all the eye muscles, interior as well as exterior, are thus affected. The eyeball in this case is fixed and motionless, so that the condition closely resembles a total ophthalmoplegia, from which, indeed, it can be differentiated only by the fact that there are miosis and spasm of accommodation.

Spasm also is occasionally due to *hysteria*. In most of the cases, however, in which the spasm is reported as affecting the eye muscles, it really affected some one of the co-ordinate movements of the eyes. In particular, the cases of spasm of the interni that have been reported are probably really cases of spasm of convergence. The same is probably true of Theobald's case of spastic squint occurring after *influenza*.

The spasm that *accompanies convulsive attacks* is also usually a conjugate spasm, affecting the associated movements rather than a single muscle. Gowers, however, records several cases in which a single muscle, especially the external rectus, was the seat of a true epileptoid attack,—*i.e.*, the muscle was suddenly seized with tonic or combined tonic and clonic spasms, which, after lasting a short time, disappeared, leaving the eye completely normal until the next attack. The seizures were sometimes preceded by an aura and were accompanied by vertigo, diplopia, and some obscuration of consciousness.

Gowers also states that slight clonic spasms of the eye muscles may occur in *chorea*.

Desmarres, the celebrated ophthalmologist, suffered from a clonic spasm of the superior oblique, due to a lesion of the opposite cerebral hemisphere (Landolt, cited by A. Graefe).

Secondary spasm is much more common than the prim ondary spasm we mean one which is the result of a paralysis of another muscle, either in the same eye or in its fellow. It occurs under the following forms:

1. *Secondary spasmodic deviation of the sound eye when the paretic eye fixes* (page 215). This deviation, as already shown (page 217 and *cf.* Table, page 247), always appears under the guise of a spasm of the associate to the paralyzed muscle.

2. *Spasm of a synergic muscle in the same eye.* If the right superior rectus is paralyzed, the extra nervous effort employed in keeping this eye in place will be imparted equally to its synergic muscle, the right inferior oblique. This muscle, in consequence, acts excessively. In most cases the effect of this excessive action is not apparent, because the associate muscle in the left eye (the superior rectus) receives a similar excessive impulse. Sometimes, however, the impulse imparted to the oblique preponderates, and then we have the picture of a combined paralysis of the right superior rectus and spasm of the right inferior oblique. That is, we have a left diplopia which increases in looking up and to the right, and a right diplopia which increases up and to the left. This finding is quite like that which we get in a paralysis of both superior recti, and often differs only in the fact that the diplopia in the left upper field fluctuates markedly in amount from one examination to another.

In an abducens paralysis we sometimes find a similar associated spasm of the obliques. This causes the eye to move by zig-zag jumps a little further out than the externus alone can carry it. If the eye is above or below the horizontal plane, the amount of additional movement thus obtained may be considerable.

3. More or less irregular and varying *spasm in the opponents of the paralyzed muscle* (page 244).

4. *Permanent spastic contraction* (*contracture*) *of the opponent* to the paralyzed muscle (page 243). As already noted, in this case the paralysis itself may disappear and the contracture remain. When this happens the picture presented will be that of spasm, pure and simple.

SECTION IV.

CONVERGENCE AND DIVERGENCE ANOMALIES. COMITANT HETEROPHORIA AND SQUINT.

Nature of these Anomalies.—The remaining motor anomalies of the eye are not caused by weakness or overaction of the muscles, *per se,* nor by any affection of the nerves supplying them. They are due to involvement of the centres or tracts connecting these nerves with one another and with the cortex, and consequently appear under the guise of either a deficiency or an excess of one of the co-ordinated movements of the eyes (convergence, divergence, or parallel movement).

Convergence, divergence, and parallel movements are binocular acts,—*i.e.*, each eye takes a precisely equal part in executing them. Hence, if there is any excess or deficiency in the performance of these movements, owing to involvement of the centres governing them, the excess or the defect will show itself in each eye equally. Perversions of these functions are *always binocular*.

When we look to the right we converge or diverge our eyes with the same facility as when we look to the left, and with nearly the same facility as when we look straight ahead. The deviation produced by a convergence or divergence anomaly, therefore, will remain about the same, no matter how the eyes are turned, provided that the object of fixation is kept at the same distance from the eyes. But since generally we use our convergence strongly when looking at near objects and do not use it when looking at distance, the effect of a convergence anomaly in producing deviations will be much greater in near than in distant vision. The opposite will be the case with a divergence anomaly. *Convergence and divergence anomalies then are comitant in that the deviation changes but little* as *the eyes are carried to the right or left, and are periodic in that the deviation due to a convergence anomaly increases, and that due to a divergence anomaly decreases, as the object of fixation approaches the eyes.*

Nearly all cases of periodic comitant heterophoria and squint are produced by an anomaly of convergence or divergence. Nearly all cases of continuous comitant squint are produced by an anomaly of convergence combined with one of divergence.

Convergence-excess will produce an active, and divergence-insufficiency a passive, convergence of the visual lines (esophoria or esotropia). Convergence-insufficiency and divergence-excess will produce exophoria and exotropia. Ordinary comitant convergent squint (esotropia) is due to a convergence-excess combined with a divergence-insufficiency, and ordinary divergent squint (exotropia) to divergence-excess combined with convergence-insufficiency.

Types of Periodic and Continuous Deviations.—The different types of periodic and continuous deviations thus produced have the following characters:

PERIODIC HETEROPHORIA AND SQUINT

	ESOPHORIA AND CONVERGENT SQUINT		EXOPHORIA AND DIVERGENT SQUINT	
	CONVERGENCE-EXCESS	DIVERGENCE-INSUFFICIENCY	CONVERGENCE-INSUFFICIENCY	DIVERGENCE-EXCESS
Deviation for distance by all tests (screen, parallax, Maddox rod, phorometer).	Orthophoria or slight esophoria.	Esophoria marked.	Orthophoria or slight exophoria.	Exophoria marked.
Deviation for near (by all tests).	Esophoria marked (much more than for distance).	Esophoria slight or none (decidedly less than for distance).	Exophoria very marked (much more than for distance).	Exophoria decidedly less than for distance.
Convergence near-point.	Normal (or abnormally close).	Normal.	Remote (more than 7 cm. from root of nose).	Normal.
Prism-convergence (adduction.)	Normal.	Normal.	Low and accomplished only with effort and difficulty.	Normal.
Prism-divergence (abduction.)	Normal.	Low less than (2°-3°) or negative (*i.e.*, insuperable homonymous diplopia for distance).	Normal.	Abnormally great (over 9° of prism).
Primary and secondary screen deviation.	In all four types deviation of one eye behind the screen, measured with prisms, equals precisely that of the other eye (distinction from paretic deviations).*			
Field of binocular fixation.	In all four types excursions of each eye normal in all directions. Neither eye lags behind the other when both are making lateral, vertical, or oblique movements (distinction from paretic deviations).			
Field of binocular single vision.	Homonymous diplopia if present, not increasing (if anything, diminishing) as eyes are carried to right and left (distinction from paretic deviations). Increases somewhat in looking down, diminishes in looking up.		Crossed diplopia not increasing as eyes are carried to right and left (distinction from paretic deviations). Increases somewhat in looking up, diminishes in looking down.	

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*In some few cases one eye does deviate more than the other when covered. This seems to occur in old cases of squint, in which secondary muscular changes have taken place in the squinting eye, so that the latter has in effect either a paretic or an overacting musculature. It also happens when, owing to excess of hyperopia in one eye or to some similar cause, the patient exerts more accommodative effort when seeing with one eye than when seeing with the other. If, for instance, a man's right eye is much more hyperopic than his left he will accommodate strongly when the right fixes and the left is screened, and hence the left will then turn strongly in. Then when the screen is shifted to the right eye and the left fixes, less accommodative effort will be put forth, and the right eye will turn in but little or may even under certain conditions turn out (Graefe).

CONTINUOUS HETEROPHORIA AND SQUINT

	ESOPHORIA AND CONVERGENT SQUINT =convergence-excess + divergence-insufficiency.	EXOPHORIA AND DIVERGENT SQUINT =convergence-insufficiency+divergence-excess.
Deviation for distance and near (by all tests), but especially the screen.	Very marked esophoria or a marked convergent squint at all ranges (somewhat greater for near than for distance if the convergence-excess was the primary condition, and greater for distance than for near, if the divergence-insufficiency was the primary condition).	Marked exophoria or marked divergent squint at all ranges (somewhat greater for near than for distance if the convergence-insufficiency was the primary condition, and greater for distance than for near if the divergence-excess was the primary condition).
Convergence near-point.	Normal or excessively close.	Remote. In case of actual squint, none (patient may try to converge but cannot secure binocular fixation at any distance).
Prism convergence (adduction).	If it can be elicited (*i.e.*, if there is no suppression), normal or supernormal.	Low. In case of actual squint negative (*i.e.*, there is either crossed diplopia at all ranges so that no prism is overcome, or there is monocular vision with suppression).
Prism divergence (abduction).	Low. Usually (in case of squint, always) negative (*i.e.*, there is homonymous diplopia at all ranges, so that no prism, base in, is overcome, or there is monocular vision with suppression).	If it can be elicited (*i.e.*, if there is no suppression) it is much greater than normal.
Primary and secondary screen deviation.	As in periodic deviations. (See table preceding.)	As in periodic deviations. (See table preceding.)
Field of binocular fixation.	As in periodic deviations. (See table preceding.)	As in periodic deviations. (See table preceding.)
Field of binocular single vision.	As in periodic deviations. (See table preceding.)	As in periodic deviations. (See table preceding.)

Esophoria and the Development of Convergent Squint.—1. Development of Squint from a Convergence-excess.—A convergence-excess, as shown in the table, has the following characters:

Esophoria or evidences of inward deviation by all tests, pronounced for near, slight, or absent for distance; convergence near-point and prism-convergence normal; prism-divergence normal or nearly so; inward and outward excursions of the eyes normal, deviation not increasing to either right or left.

Convergence-excess is nearly always *accommodative,* being due to the excessive effort which the patient makes to overcome hyperopia or astigmatism. An effort of accommodation regularly implies a corresponding effort of convergence, so that as he strains one he also strains the other.

A similar accommodative convergence-excess may be produced temporarily by the instillation of atropine or homatropine. The patient in this case tries extremely hard to use his failing accommodation, and makes the effort to do so even when his accommodation is paralyzed. To this effort his accommodation cannot respond, but his convergence does so, producing a temporary marked deviation.

A similar variety of convergence-excess, due to the attempt to employ a subnormal accommodative power, occurs when the eyes are used too soon after debilitating diseases, as measles, etc.

A convergence-excess may also be caused by an attempt to see clearly in conditions of difficult vision due to opacities of the media, low degrees of astigmatism, or even a slight amount of myopia.

In some cases of accommodative convergence-excess the peculiar conditions present cause an esophoria that, contrary to the general rule, is more marked for distance than for near. Thus, an adult with a high amount of hyperopia may succeed in overcoming it for distance, but in overcoming it may use so much accommodation as to produce also a considerable degree of convergence, while for near-points he may be utterly unable to maintain the necessary accommodation, and hence neither accommodates nor converges.

Occasionally a convergence-excess is met with in which *no accommodative element* is present.

The *use of prisms, base out,* especially for constant wear, often produce a convergence-excess or decidedly aggravates one already present.

Cases of true *spasm of convergence* (spastic contraction of the interni producing a marked convergent squint) are sometimes seen, especially in hysteria (Hitzig cited by Graefe; Fuchs).

A convergence-excess when moderate *may disappear.* This, for instance, happens in adults who, because of their advancing years, no longer exert enough accommodation to overcome their hyperopia, and hence give up the effort and with it the excessive convergence.

In other cases a convergence-excess, especially when it occurs in childhood and when of high degree, tends to increase and ultimately *develops into a convergent squint.* The steps by which this occurs are as follows:

(1) A child with a good deal of hyperopia or astigmatism, as soon as he begins to make much use of the eyes for near work, develops, as a result of undue accommodation, a very varying, evidently more or

less *spasmodic esophoria*, which is marked for near when his convergence is called into play, but which is little or not at all marked for distance. The result is either asthenopia, due to the troublesome effort to overcome the deviation, or else a confusing condition of alternating diplopia and single vision. Usually at this period there is still binocular fixation even for near, although every now and then when the convergence is particularly called upon (for instance, when a pencil at which the eyes are directed is carried quickly toward the nose), one of the eyes may turn suddenly and sharply in, producing a *momentary spasmodic squint*.

(2) Little by little the deviation increases until binocular fixation for near becomes impossible, and the periodic esophoria is transformed into a *periodic convergent squint*. This at first is intermittent and occasional, afterwards more and more constant. It will now be found that there is more or less esophoria for distance also, although always less than for near. There is still, however, binocular fixation for distance.

Careful tests at this stage will show in nearly every case that *the squint is associated with diplopia*. This diplopia which presents sudden spontaneous variations in amount from one moment to another, and which also varies with the distance of the test-object from the eyes, is evidently a source of not a little trouble and confusion.

(3) After this condition has lasted for a time, perhaps for some years, the patient gives up binocular fixation for distance, so that he now has a convergent squint for both distance and near (*continuous squint*). The deviation as measured by the screen, however, is still much more marked for near than for distance. Diplopia can still usually be elicited by the tests. The condition is still mainly one of convergence-excess.

(4) Later still, the amount of deviation for distance begins to increase until ultimately the squint for distance equals that for near-points, and both are absolutely greater than they were. This change is due to the superadding of a divergence-insufficiency to the primitive convergence-excess; the conditions now, in fact, are those shown in the table as obtaining for *a convergence-excess and divergence-insufficiency combined*. The rotations of the eyes are still normal in amount, indicating that the muscles themselves are as yet unaffected.

At this stage, diplopia, if it can be elicited at all, no longer obtrudes itself upon the patient's notice or causes confusion. In many cases it cannot be elicited by any tests, suppression of one of the images having occurred (page 187).

(5) Later, *secondary muscular changes* (contracture of the internus, stretching and weakening of the externus) take place in the squinting eye, producing an absolute reduction in the rotation outward and excess of rotation inward. When this occurs we can speak of the squint as being in a sense monocular, but until then it must be regarded as the result of a strictly binocular process.

2. **Development of Convergent Squint from a Divergence-insufficiency.**—A **divergence-insufficiency**, as shown in the table, has the following characters:

Marked esophoria and very low diverging power, or, often indeed, insuperable or varying homonymous diplopia for distance; normal or nearly normal relations for near; no impairment of outward nor excess of inward rotations of the eyes.

As a primary affection it is rather rare, although I have seen several well-marked instances. Often, especially when slight, it seems to remain unchanged indefinitely; in other cases it may increase, producing a convergent squint. This it does apparently first by the production of an increase of the esophoria for distance, then by the superadding of a convergence-excess, producing an esophoria for near; the result is, first a periodic convergent squint (esotropia for distance, esophoria with binocular fixation for near); later a continuous squint for all ranges. Until late in their course such cases are distinguished from those starting with a primitive convergence-excess by the fact that the deviation for distance always exceeds that for near.

Some cases of convergent squint develop suddenly under the guise of a **divergence-paralysis.** This must be regarded simply as an extreme form of a divergence-insufficiency in which the diverging power is not simply diminished but abolished. It is characterized by homonymous diplopia, which is very great in amount for distance and diminishes with mathematical regularity as the test-object is brought toward the eyes, and which, instead of growing greater, tends to grow less as the eyes are directed to the right or to the left; marked esotropia for distance diminishing progressively as the object of vision is carried toward the eyes and finally changing to orthophoria at some near point; no restriction in the movement of either externus or excess of action of either internus, so that the excursions of the eyes are normal. Vertigo is often marked and there may be false projection.

Divergence-paralysis usually develops suddenly. In some seven of the reported cases, including two of my own, organic cerebral lesion appeared to cause the difficulty, there being a history of syphilis, tabes, multiple sclerosis, lead-poisoning, or the evidences of cerebral tumor.

Another case appeared to be due to concussion of the brain, and still another to an antecedent pneumonia and pleurisy. The condition in two of Uhthoff's cases was preceded by bilateral abducens paralysis, and one of Bielschowsky's appeared to go into a unilateral paresis of the abducens. In many cases the affection occurs without obvious cause. Its duration is uncertain, some cases lasting at least four or five years, others recovering in a year or so.

Exophoria and the Development of Divergent Squint.—1. Development of Squint from a Convergence-insufficiency.—Most cases of divergent squint develop from a convergence-insufficiency. This has the following character:

Exophoria, marked for near, slight for distance and not increasing to either right or left; convergence near-point remote, being seven centimetres or more from the root of the nose; prism-convergence subnormal and acquired with difficulty; prism-divergence about normal; rotation of the eyes inward and outward, normal.

In many cases the convergence-insufficiency is *accommodative*, that is, it occurs in those who do not require to use their accommodation in the normal way. The accommodation and convergence being intimately associated, the disuse of one means the disuse of the other. This disuse of the convergence often shows itself at first simply by the presence of a large amount of exophoria for near without any recession of the convergence near-point, but later it develops into a true convergence-insufficiency.

Accommodative convergence-insufficiency occurs in myopes who are not using concave glasses, and may also occur (from sudden relaxation of the accommodation) in hyperopes and presbyopes when putting on convex glasses for the first time. A somewhat similar relaxation of the convergence is found in those who for any reason (presbyopia, marked hyperopia, anisometropia, etc.) do not see well for near; and in this case the convergence-insufficiency may be relieved by convex glasses.

A *non-accommodative* convergence-insufficiency,—*i.e.*, one which develops without any reference to a refractive error,—occurs quite often in hysteria, traumatic neuroses, neurasthenia, anæmia, and debilitated conditions, however caused; also in connection with intranasal disease, and not infrequently seems to occur in those who are apparently healthy. According to Guillery it may be produced by poisons (alcohol, ether, chloroform, and chloral).

A convergence-insufficiency, especially if slight, may remain unchanged indefinitely; in other cases, particularly if of large amount, it *develops into a divergent squint.* The successive stages of the process will then be as follows:

(1) Marked exophoria for near with at first a normal near-point of convergence; orthophoria for distance and normal diverging power (*periodic exophoria*).

(2) Beginning divergent squint for near with recession of the convergence near-point; beginning exophoria for distance with increase in the prism-divergence (*periodic divergent squint*). This squint, at first intermittent, is afterwards constant.

(3) Marked increase of the exophoria for distance with gradual transformation into squint (consecutive divergence-excess) converting the periodic into a *continuous squint.*

(4) *Muscular changes* (contracture of the externus, stretching and weakening of the internus) producing absolute impairment of rotation inward and excess of rotation outward.

In some cases a periodic divergent squint develops suddenly from a **convergence-paralysis.** This is characterized by crossed diplopia and exotropia, which both increase with mathematical regularity as the test-object is carried towards the eye. The deviation and the diplopia do not increase either to the right or to the left. Movements of each eye outward and inward are normal.

A peculiar symptom of convergence-paralysis is the *absence of the convergence-reaction* of the pupil. If, as is usually the case, the light reaction is present, the appearance presented is the converse of the Argyll-Robertson pupil. Accommodation is sometimes diminished or absent, sometimes normal.

Convergence-paralysis often develops suddenly and is frequently associated with very marked vertigo (Parinaud) and with more or less false projection (M. Sachs). It is not very infrequent, occurring in tabes, multiple sclerosis (in 3 per cent. of the cases), and probably as a result of other organic nervous diseases. It is sometimes associated with paralysis of divergence or with paralysis of up and down movements (Parinaud). I have seen a marked instance of the former combination in a woman who, at a distance of twenty inches, had orthophoria and, an inch or two further out, homonymous diplopia, increasing *pari passu* as the object of vision was carried off, while within twenty inches she had crossed diplopia, increasing fast as the object was carried towards the eyes.

A similar absence of divergence and convergence power, constituting what A. Graefe calls *diminution in the amplitude of fusion,* is found in multiple sclerosis (Uhthoff) and paralysis of the ocular muscles due to central disease (Soelberg-Wells). This condition is marked by the fact that the patient cannot overcome any amount of diplopia, however minute, produced by prisms. Such inability to overcome prisms, however, may also be due to sudden failure of the fusion faculty (p. 185).

Convergence-paralysis may get well spontaneously or remain indefinitely.

The anatomical site of the lesion is probably in the fibres connecting the non-decussating fasciculi of the two internal recti nuclei with each other, or connecting these fasciculi with the cortex (Bernheimer); or it may be cortical.

2. **Divergent Squint developing from a Divergence-excess.**—The characters of a *divergence-excess* are as follows:

Exophoria by all tests, pronounced for distance, slight or absent for near; near-point of convergence and prism-convergence normal; prism-divergence excessive, being over 9°; movements of eyes inwards and outwards normal; deviation not increasing to either right or left.

This condition often remains unchanged for a long period, even for years. In not a few instances, however, it develops into a divergent squint. The steps in the process in this case are as follows:

(1) Marked exophoria for distance with excessive prism-divergence; exophoria for near, little or none, and near-point of convergence normal (*periodic exophoria*).

(2) Beginning divergent squint for distance with crossed diplopia and still further increase in the prism-divergence; beginning exophoria for near, but with convergence near-point still normal (*periodic divergent squint*). This squint is at first intermittent, afterwards constant.

(3) Marked increase of the exophoria for near and gradual recession of the convergence near-point (*consecutive convergence-insufficiency*), producing a squint for near, which, however, is less than for distance).

(4) Marked divergent squint which is of about the same amount for both distance and near (*conversion of the periodic into a continuous squint by the superadding of a convergence-insufficiency to the primitive divergence-excess*). Diplopia still often present, but now usually negligible by the patient. The rotations of the eye inward and outward are still about normal. The patient may still make strong converging efforts, so that though he has no longer an absolute he has still a relative convergence near-point.

(5) *Muscular changes* (weakening of the interni; contracture of the externi) causing impairment of rotation inwards and excess of rotation outwards.

Rationale of the Development of Squint.—**How and when Squint develops.**—A squint in most cases apparently arises from an instinctive endeavor to avoid diplopia and confusing images. It seems quite certain that *in their inception the different periodic deviations are accompanied by diplopia.* If the deviation is slight the patient overcomes the double vision with ease; if it is great, with more and more difficulty. In the latter case ultimately he gives up the effort to overcome the diplopia and then sees double all the time.

Now it becomes apparent when we study these cases that the more nearly continuous (non-periodic) the deviation is the easier it is for the patient to ignore the diplopia to which it gives rise, and the less pronounced, consequently, are the subjective disturbances which are dependent on this diplopia.

Again, it is almost always the case that a large deviation causes much less subjective trouble than does a small one: first, because the image projected by the deviating eye is so faint and so far from the other that the patient can readily ignore it; second, because the deviation being so large that the patient cannot possibly overcome it, he does not even make the attempt to do so, and thus escapes the reflex disturbance that such an attempt often produces. Accordingly, it is for the patient's interest that *if he has a periodic deviation it shall be transformed into one that is as nearly continuous as possible;* and, second, *that if it is small it shall increase in amount until the diplopia is readily negligible.*

As we have just seen, both kinds of change occur regularly in the development of heterophoria, and particularly in the evolution of convergent and divergent squint.

The processes by which these changes take place are not located in the muscles themselves, for until very late in the development of most cases of squint the rotations of the eyes are normal, indicating that the power of the muscles *per se* is neither impaired nor excessive. The *changes that take place are doubtless central,* following, as Hoffmann pointed out, the law promulgated by Sherrington, that excessive stimulation of the centre for one movement produces inhibition of the centre for the opposing movement, and *vice versa.* In other words, in cases of convergence-excess the centre for convergent movements being subjected to continuous overstimulation, becomes more and more active, producing a continually greater excess of convergence action and at the same time causing greater and greater inhibition of the centre for the divergent movements. The consequence is that superadded to the convergence-excess we have ultimately a progressive divergence-insufficiency. It is only late in the course of the condition that the persistent stimulation of the internus, coupled with the maintenance of one eye in the forced inward position, produces hypertrophy or contracture of this muscle and stretching or atrophy of its opponent.

A small proportion of cases of squint do, however, *develop out of a true muscular defect*. Thus, some cases of convergent squint, both in childhood and in adult life, develop out of a primitive paralysis of the abducens which has been converted into a comitant deviation by consecutive overaction and contracture of the internus (page 243). Furthermore, a convergent squint due to a convergence-excess may be aggravated by the presence of a primitive weakness of the externi or overdevelopment of the interni. Again, a divergent squint may be produced by developmental changes in the orbit causing a relative preponderance of the externi (page 221), or by weakness of the internus due to operation. Such factors, however, play a very subordinate part in the production of squint.

Conditions favoring the Development of Squint; Period at which it develops.—The development of a convergent squint out of a heterophoria in the way just outlined is much more apt to occur if there is any condition such as anisometropia, opacities of the media, fundus disease, congenital anomalies, etc., that *renders the sight of one eye much worse than that of the other*. In such a case the image of the worse eye often confuses that of its fellow instead of reinforcing it, and hence the patient really sees better when he allows the worse eye to deviate. In many cases it is apparent that he actually forces it to deviate so as to get the disturbing image out of the way. This is particularly the case in divergent squint.

A factor which may be of considerable importance and which, indeed, by many is regarded as the predominating element in the production of squint is the *lack of development of the fusion-faculty* (page 185). The effect of this would be that the patient would have no tendency to blend the two retinal images, and hence would have no special motive for maintaining binocular fixation. Consequently, if there was a deviation of the eyes, he would not try to overcome it, but would allow one of them to squint.

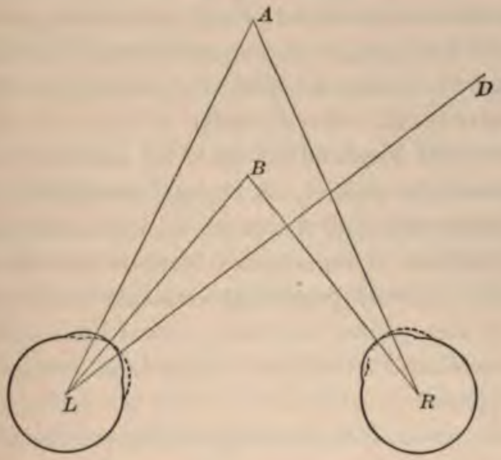
Ill-development of the fusion-faculty and the squint that it produces may be hereditary. Some, indeed, go so far as to regard *heredity* as the main factor in the genesis of squint. This view, I think, is not substantiated by the facts.

A *convergent squint* usually develops in early childhood. Once developed, it increases to a certain point, then generally remains unchanged through life. In not a few cases, however, it diminishes in later childhood, and may even disappear altogether or be converted into a divergent squint. These changes are probably due to developmental processes (divergence of the orbits during the growing period), which tend to render the power of the externi greater and that of the interni less (see Weiss, page 221).

Squint develops very often in congenital spastic paralysis (Little's disease).

Divergent squint generally occurs later in life and is more often accompanied by persistent diplopia than is convergent squint. It sometimes develops *after a tenotomy done for convergent squint,* and this may occur even years after the operation. This secondary exotropia is traceable sometimes to an insufficiency of the internus produced by the tenotomy; more often it is not attributable to the latter, being due either to the developmental changes (divergence of the orbits) above mentioned, or to the supervention of a true divergence-excess.

FIG. 15.



Convergent squint occurs about three times as often as divergent squint.* In anisometropia, however, divergent squint is the more frequent form.

Squint a Bilateral Affection.—In squint the patient appears simply to fix with one eye and to turn the other inwards or outwards. In reality, however, both eyes are turned. Thus, as shown in Fig. 15, when a patient with convergent squint is told to look at the object A, both eyes tend to converge equally, so that they really look at some nearer point, like B; then, in order to get the image of A on the fovea of at least one eye, the patient turns both eyes to one side—say to the right—through the equal angles BRA and BLD. The right eye, R, is now directed towards A or fixes it, the left eye squints in by the angle ALD=2×BLD. Thus, here really the squint is bilateral, each eye actually turning in by an equal amount, yet apparently the left eye alone squints, because the patient happens to use the right for fixation.

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* The disproportion is greater than this if we judge from most hospital statistics, but this probably arises from the fact that slight degrees of divergent squint are frequently overlooked.

Which eye the patient actually uses to fix with depends usually upon the vision. If he sees much better with one eye than with the other he will fix with the better and squint with the worse eye; the squint will then be unilateral. If the two eyes are about alike in vision and refracting power, we often find an alternating squint, the patient using either eye indifferently for fixation and squinting with the other. In some cases, particularly in anisometropia, the patient may fix with one eye for distance and with the other for near.

Hyperphoria and Vertical Squint.—Hyperphoria in a great many instances is caused by paresis, or at least insufficiency of one of the elevator or depressor muscles of the eye. In this case the deviation and its evidences (vertical diplopia, etc.) increase and decrease characteristically in looking in some particular direction of the gaze (page 215). Hyperphoria of high degree and real vertical squint (hypertropia and hypotropia) are almost always of this origin, being, therefore, *non-comitant* deviations.

In some cases, even when thus originating, a hyperphoria or vertical squint may become nearly comitant by virtue of consecutive contracture of the opponents of the paretic muscles (page 243).

In a few instances even a very high degree of hyperphoria or a true vertical squint may be truly *comitant* from the start,—*i.e.*, we can find no evidence of insufficiency of any elevator or depressor muscle. Hyperphorias of low degree are usually comitant.

Some cases of hyperphoria seem to be *spasmodic* in origin, changing rapidly and lasting only a short time. Sometimes a spasmodic hyperphoria of this sort seems to be due in some way to accommodative action, since it disappears apparently as the result of the correction of a refractive error. In most cases hyperphoria once developed lasts indefinitely. It does not, as a rule, tend to increase, except in the form due to marked paralysis of an elevator or depressor where the deviation is often exaggerated as time goes on, either by secondary contractures of the opposing muscles or by a secondary deviation in the good eye due to fixation with the one that is paretic (page 215).

Symptoms of Heterophoria and Squint.—These are—

1. **Diplopia.**—This, especially when slight so that the double images are close enough together to confuse each other, produces blurring of sight and sometimes vertigo.

2. **Reflex Symptoms induced by the Effort made to correct the Deviation.**—These are asthenopia, pain in the eyes, conjunctival irritation, headache, which may be supraorbital, orbital, temporal, or occipital, and other pains situated in various parts of the body. Occasionally, also, we find spasm in the facial muscles, and sometimes other forms of spasm; not infrequently also digestive disturbances, nausea, and interference with the general nutrition.

Reflex symptoms are especially pronounced when the deviation is so *slight* that the patient can overcome it, and consequently does so, although with effort. They are particularly marked when the deviation is inconstant and varies under different conditions, because in this case the patient cannot as readily allow for the changing position of the eyes as he can when the deviation is the same all the time. Consequently, reflex disturbances are *greater in heterophoria* than they are in squint and *greater in a periodic squint* than they are in one which is continuous. Indeed, as we have seen, this very fact seems to constitute the reason why a periodic is regularly converted into a continuous squint.

Both the tendency to diplopia and the reflex disturbances caused by muscular anomalies increase in proportion to the difficulty that the patient meets with in maintaining binocular fixation. They are hence *greater under conditions demanding very accurate and very sustained fixation,*—greater, for instance, when the patient is doing continuous fine work or when he is looking steadily at brightly illuminated or sharply defined objects, particularly when such objects are in motion. Hence, many persons with moderate muscular anomalies always suffer from headache and fatigue (panorama headache, panorama asthenopia) after shopping or going to the theatre, or after seeing a ball-match or a street-parade.

The most marked and disagreeable symptoms are found in hyperphoria and in divergence-insufficiency. In these conditions the symptoms are usually most troublesome in *distant vision.*

In convergence-insufficiency the commonest symptoms are asthenopia, headache, and conjunctival irritation, and these are developed generally by *near work.* They are much more apt to [illegible] the non-accommodative than in the accommodative form of convergence-insufficiency.

Treatment of Heterophoria and Squint.—1. **Correction of Refraction.**—This is a very important element in all of these anomalies and may alone suffice to effect a cure, particularly when the deviation is *still of the periodic type.* It is of special importance and efficacy in convergence-excess, particularly convergence-excess which is developing into squint. In this condition it is necessary to correct the whole of the hyperopia and astigmatism present. Similarly, in convergence-insufficiency accompanying myopia, it is of great importance to make the patient wear a glass fully correcting his error and for near as well as for distance.

In cases in which the deviation is *no longer periodic,—i.e.,* in which, in addition to a convergence anomaly, there is superadded an anomaly of divergence,—correction of the refraction is less apt to afford a cure, and in the case of a *continuous squint* very rarely does more than diminish the amount of the deviation. In any case, the effect of correction of refraction is *rarely an immediate one;* it usually takes some months—perhaps a year or two—for the full effect of the glasses to be secured.

2. **General treatment** must be employed in neurasthenia and hysteria and in conditions of debility from whatever cause. This will often relieve the symptoms without itself producing any material change in the muscular condition of the eyes.

3. **Treatment of Associated Conditions.**—The treatment of other affections, particularly intranasal disease, which may possibly be sources of reflex trouble, should never be neglected.

4. **Exercises in moving the Eyes.**—Exercises of the eyes in converging on a pencil carried towards the nose or in overcoming prisms, base out, are useful in convergence-insufficiency and occasionally in divergence-excess.

5. **Orthoptic Exercises.**—With a squint that develops in early life much can be done by educating the fusion faculty. This can be done with the *stereoscope,* or, better still, with Worth's *amblyoscope* (Fig. 8). Both of these instruments, by compelling the eyes to act together, train the fusion-faculty. Both are at first adjusted for the patient's angle of squint, then by successive adjustments the patient is gradually taught to look into the instruments with the eyes more and more nearly straight and yet maintaining fusion. In this way the angle of squint is steadily diminished, and there is finally orthophoria.

Another orthoptic exercise is *bar-reading.* This consists in reading with a bar held between the eyes and the print. Unless the patient is using both eyes at once for seeing, the bar will hide some of the letters. The use of the bar is an exercise in binocular vision.

The squinting eye can also be exercised either by *bandaging the good eye*, or, better still, by keeping the latter for some weeks under *atropine*.

6. **Prisms for Wear.**—Deviations of low degree may be corrected by prisms worn constantly, either alone or combined with the glass correcting the refraction. This is most serviceable in hyperphoria. In esophoria and exophoria, prisms constantly worn often tend to increase the deviation, and their use is not generally advisable. Moreover, it is not usually possible for a patient to wear prisms of 3°, or at most 4°, before one eye, so that the total amount of deviation that we can correct by this means would not be more than 7° at most.

7. **Operation.**—When everything else has been tried and has failed, an operation is indicated.

With an *esophoria* or *convergent squint*, tenotomy of one or, better, of both internal recti is done, provided the condition is mainly one of convergence-excess. When the condition is one of divergence-insufficiency, the better plan is advancement of one or both externi.

Similarly, in *exophoria* or *divergent squint* caused by divergence-excess, tenotomy of one or, better, of both external recti, is indicated. Cases of convergence-insufficiency, on the other hand, should be treated by advancement of the interni. In marked divergent strabismus tenotomy of one or both externi, combined with advancement of the interni, usually has to be done.

In *hyperphoria*, if non-comitant, we proceed according to the rules laid down for operative interference in paretic deviations (page 245). In a comitant hyperphoria the best operation is a tenotomy of the superior rectus, which should never be pushed so far as to render the muscle paretic nor so far as to produce even a moderate overcorrection.

SECTION V.

DERANGEMENTS OF THE ASSOCIATED PARALLEL MOVEMENTS OF THE EYES.

The derangements of the associated parallel movements of the ey comprise—

A. Paralysis and spasm of the parallel movements (conjug paralysis, conjugate spasm).

B. Tremor and ataxia of parallel movements (nystagmus, pse nystagmus).

Conjugate Paralysis and Spasm.

Nature of these Anomalies.—We not infrequently meet with cases in which the ability of the eyes to perform some parallel movement is impaired or abolished (**conjugate paralysis**). Thus, both eyes may move from left to right as far as the middle line, and then stop short, refusing to go any farther (paralysis of dextroversion). In this case commonly the two eyes and sometimes also the head are deviated in the opposite direction,*—*i.e.*, show a *conjugate deviation* to the left. This conjugate deviation in a paralysis of the right-rotators is caused by an overaction of the antagonists, or left-rotators. Such overaction is probably central in origin, since Sherrington's experiments seem to indicate that abolition of function in the nerve-centre presiding over any movement is regularly associated with exaltation of function in the centre that presides over the opposing movement.

In some cases conjugate deviation is due not to a conjugate paralysis, but to a **conjugate spasm**. For example, we may find both eyes deviated to the left, not because the right-rotators are paralyzed, but because the left-rotators are in a state of primary spastic contraction. Such a conjugate spasm results from an irritative lesion of the centre for left-rotation, just as a conjugate paralysis results from a destructive or inhibiting lesion of the centre for right rotation. It is distinguished from paralysis by the fact that, although strongly deviated in one direction, the eyes can usually be moved more or less freely in the direction opposite.

Symptoms.—Since the deviation is alike in the two eyes, conjugate paralysis and spasm do not cause diplopia. They may, however, cause *vertigo* and *false projection* (A. Graefe; M. Sachs). In a conjugate deviation to the left, due to paralysis, the patient would overshoot the mark when trying to touch an object situated on his right, while if the deviation were due to spasm he would undershoot the mark when trying to touch an object situated on his left.

Very frequently in cases of conjugate deviation any symptoms that may be occasioned by the eye trouble are masked by the grave general symptoms due to the causal disease.

Course.—Conjugate paralysis occurs in all degrees of *intensity*, being sometimes very slight, sometimes complete. It usually develops suddenly, and is often transient. The conjugate deviation which the paralysis produces often disappears long before the paralysis itself. This is especially true of cerebral disease. Here the conjugate deviation, which is quite pronounced during the period of coma, often disappears within a few hours, and rarely lasts more than three or four weeks, although when it does go it generally still leaves a partial conjugate paralysis behind.

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* In rare cases the head may be deviated in one direction and the eyes in the direction opposite (*alternating conjugate deviation* of Bresler).

Non-paralytic Restriction of Conjugate Movements; Diagnosis from True Paralysis.—A conjugate paralysis must be differentiated from the condition in which the patient can move the eyes in the given direction, but does not move them, either from wish to deceive or from fear of pain, or because he really does not know how to make the effort. In some cases, according to Gunn, the patient, when told to look to one side, or up, or down, seems unable to initiate the movement of his own will, although he can follow a slowly-moving object that is carried in the given direction. In other cases he cannot even do this, and yet the power of movement, although apparently never exerted, is really there, as we can prove by *Graefe's test.* This test is best performed by making the patient look at a distant object, and then placing a prism of 10° or 20° before each eye, with the apex directed in the way in which we wish the eyes to move (*e.g.*, to the right in paralysis of dextroversion, etc.). If the patient really has a conjugate paralysis, his eyes will move neither when the prisms are put before them nor when they are taken away. If, however, he can move the eyes, we shall see each make a distinct jump in the direction of the prism-apex when the prisms are put on, and a distinct jump in the opposite direction when they are taken off.

More or less allied to conjugate paralysis is the curious condition of *inertia of the ocular movements* described by Gowers as occurring in a case of progressive muscular atrophy. The patient when gazing at one object was told to look at another situated 45° to the right or left. He turned his head so as to front the second object, but kept his eyes still fixed on the first, and only after a time, and slowly then, transferred his gaze from this object to the one he was told to look at.

Varieties of Conjugate Paralysis and Spasm.—1. **Paralysis of Side-to-Side Movements (Lateriversion).**—Paralysis of a side-to-side movement (*e.g.*, of the conjugate movement to the right) occurs under the following forms:

(A) Neither eye, when attempting to follow an object that is moving to the right, can move much or at all beyond the middle line (paralysis of dextroversion, right and left). But the left eye can move to the right when converging on an object which is brought in towards the eyes in the median plane (retention of convergence). The condition here is, then, a pure paralysis of lateriversion, and the appearances it presents are, it will be seen, the precise opposite of those that obtain in a paralysis of convergence (page 257).

(B) Neither eye can follow an object that is carried to the right, provided both eyes are open, but the left eye can follow the object to the right if the right eye is covered. This is really the same case as A, the movement inwards of the left eye when the right eye is covered being simply a movement of convergence (Jeffries, Möbius).

Two unusual cases of this type have been reported in which there was paralysis of the movements of both eyes to the right, provided both eyes were open, but in which the right as well as the left eye could move to the right when separately tested. In one case (Samelsohn's) each eye could move to the right if the eye itself was screened; in the other case (that of Stevens of Denver) each eye could move to the right if the other eye was screened. In both cases the individual movement of the right eye to the right was probably a movement of active divergence (page 196) just as the movement of the left eye to the right was one of active convergence.

(C) The right eye can follow an object that is moving to the right, but the left cannot. The left eye, however, as in type A, can move to the right in performing convergence. This condition, which may be called conjugate hemiparalysis (paralysis of left dextroversion), is distinguished from a paralysis of the left internus by the fact that in the latter the left eye could move inwards neither when converging nor when attempting to make a conjugate movement to the left.

(D) Neither eye can follow an object that is carried to the right, and the left eye, moreover, cannot move to the right when attempting to converge (combined paralysis of lateriversion and convergence).

Paralysis of lateriversion is sometimes *bilateral*,—*i.e.*, both eyes fail to follow an object that is carried either to the right or to the left, although it may be that both can still converge.

2. **Spasm of lateriversion** occurs under the following forms:

(A) Both eyes are deviated spastically to one side, but can be moved freely in all directions (pure spasm of lateriversion).

(B) Both eyes are deviated spastically to one side, and cannot be moved to the side opposite (excessive spasm of one side-to-side movement, or spasm of one side-to-side movement combined with paralysis of the movement in the opposite direction).

3. **Paralysis of Up-and-Down Movements.**—Paralysis of the movement of both eyes upwards (sursumversion) or downwards (deorsumversion) is uncommon. Cases are reported by Nieden, Parinaud, Gowers, Sauvineau, A. Graefe, M. Sachs, Snell, Korniloff, Posey, etc.

It is probable that some of the cases reported are spurious, the power of movement being present but never put forth. This was so in one of my own cases, in which repeated examinations extending over several years always showed complete inability of the eyes to go upwards above the horizontal plane, and yet Graefe's test (page 267) proved that they could really turn up at least 30°, and subsequently the patient regained the power of spontaneous movement.

Paralysis of sursumversion and deorsumversion sometimes occur together, the eyes being unable to move either up or down (Lang and Fitzgerald, Wernicke, Graefe, Uhthoff), and both conditions are often associated with paralysis of convergence (in one of my cases apparently with paresis of divergence).

4. **Spasm of up or down conjugate movements** may occur, as in the case reported by Frost, in which both eyes were directed down and to the right and remained rigidly fixed in this position. Or, as in a case reported by me, there may be an intermittent clonic spasm of the oblique movements, so that the eyes at intervals darted obliquely far up and to the right and returned more slowly to the primary position. Such a condition occupies an intermediate place between true spasm and nystagmus (Compare Chapter IX).

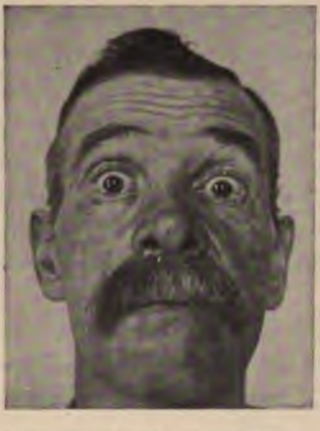

FIG. 16.—Paralysis of sursumversion and of convergence. Case of Dr. Posey.

Site of Lesion in Conjugate Paralysis and Spasm.—Paralysis and spasm of **lateriversion** may be produced by lesions in the cerebrum, cerebellum, or pons.

Cerebral lesions, especially those of sudden onset and associated with apoplectoid symptoms (coma, hemiplegia), often cause paralysis of lateriversion. A lesion of the right hemisphere produces paralysis of movements to the left, and consequently a conjugate deviation to the right. Or as Prévost puts it, the patient *turns his eyes towards the lesion.*

The lesion may be situated at various portions of the cortex, or anywhere in the subcortical association paths, or in the tracts connecting the cortex with the third and sixth nerve nuclei.

Sometimes a cerebral lesion produces a spasm of lateriversion. In this case, if situated on the right side, it will cause a spastic deviation to the left,—*i.e.,* the patient consequently *turns his eyes away from the lesion.*

According to Bernheimer, a spastic conjugate deviation, if due to cerebral disease, occurs only in cortical lesions (*e.g.,* in meningitis of the convexity of the brain).

Conjugate deviations due to cerebral disease are usually most pronounced during the period of coma, and, after this has passed off, generally disappear pretty soon—within a few days or at most a few weeks, sometimes within a few hours. Another distinguishing point is that in a paralysis of right or left movements of cerebral origin the convergence is always retained (Bernheimer), so that the picture presented is that of a pure paralysis of lateriversion (type A, page 267).

Conjugate deviation is occasionally produced by abscess of the cerebellum, the eyes being turned sometimes towards the lesion, usually away from it (Eversbusch).

Disease of the pons often causes paralysis of lateriversion, and if on the right side of the pons will produce paralysis of right-hand movements, and *vice versa,—i.e.,* the eyes *look away from the lesion.*

Small lesions in the pons involving the abducens nucleus or its immediate connections cause paralysis of lateriversion without paralysis of convergence (paralysis of types A and B, pages 267, 268). Lesions high in the pons involving only the fibres connecting the abducens and the oculo-motor nucleus produce a conjugate hemiparalysis (paralysis of the opposite internus for parallel movements but not for convergence, (type C, page 268). Large lesions in the pons above and below the abducens nucleus cause combined paralysis of lateriversion and convergence (paralysis of type D, page 268). (Jeffries, Schoeler.)

Very rarely (Schoeler; Eichhorst's case cited by Jeffries) pons disease causes a spasm of lateriversion. In this case the eyes *look towards the lesion.*

The *diagnosis between cerebral and pontine conjugate deviations* may be made from the following considerations:

CONJUGATE PARALYSIS.

In both kinds, the eyes cannot be moved in a certain direction, and usually show a well-marked deviation in the direction opposite.

CEREBRAL.	PONTINE.
Deviation transient.	Deviation more apt to be lasting.
Abducens paralyzed on same side as hemiplegia (eyes look away from paralyzed limbs).	Abducens paralyzed on side opposite to hemiplegia (eyes look towards paralyzed limbs).
The eye that cannot turn inwards in making lateral movements yet can always turn inwards in making convergence movements.	Adduction in some cases *nil* for convergence, as well as for parallel movements.
Always some associated focal symptoms.	May be none (then surely pontine).
Facial paralysis, if present, on the same side as the hemiplegia.	Involvement of the facial (usually on the side opposite to the body paralysis) very frequent. Fifth, eighth, and twelfth also often involved.

CONJUGATE SPASM.

In both, the two eyes are strongly deviated to one side, but can usually be moved more or less freely in the direction opposite.

CEREBRAL.	PONTINE.
Abducens on same side as hemiplegia spastically contracted (eyes look towards paralyzed limbs).	Abducens on side opposite to hemiplegia spastically contrasted (eyes look away from paralyzed limbs).

The following differential table furthermore will hold good for most cases:

SITE OF LESION IN CONJUGATE DEVIATIONS.

BOTH EYES ARE DEVIATED TO THE RIGHT.[*]

A. Eyes cannot be turned to the left.	B. Eyes usually can be turned to the left.
(a) There is L. hemiplegia, with or without involvement of cranial nerves on L. side.	(a) There is R. hemiplegia.
Destructive lesion of R. side of *cerebrum* or of L. side of *cerebellum*.	Irritative lesion of L. side of *cerebrum* (cortex only), or possibly of R. side of *cerebellum*.
(b) There is R. hemiplegia and usually involvement of fifth, seventh, and other cranial nerves, especially on L. side.	(b) There is L. hemiplegia with involvement of fifth, seventh, and other cranial nerves, especially on R. side.
Destructive lesion of L. side of *pons*.	Irritative or combined irritative and destructive lesion of *pons*.

Paralysis of upward movement in both eyes seems to be due usually to a lesion involving the anterior portions of both third-nerve nuclei or the corpora quadrigemina or one occupying the middle line close behind the corpora quadrigemina and affecting the gray matter about the aqueduct (Henoch, Jeffries, Gowers, Bruce). In a case with the paralysis of *both up and down movements* Wernicke found a lesion of the corpus striatum and the optic thalamus. Paralysis of up or down movements also occurs occasionally in pons lesions (in seven out of one hundred and forty cases collected by Schoeler). Judging also from Russell's and from Sherrington's experiments on animals, such a paralysis may also possibly be occasioned by a lesion of the anterior cerebral cortex.

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[*] For diagnosis of conjugate deviations to the left, substitute R. for L. and *vice versa*.

Cases simulating a Conjugate Paralysis, but of Basal or Peripheral Origin.—Conditions which closely resemble a conjugate paralysis, but which are not really such since they are not due to an affection of the centres governing conjugate movement, may be caused by symmetrical basal or peripheral lesions (*simulated conjugate paralysis*).

Thus Olbers reported a case of conjugate deviation to the right in which autopsy showed that the left internus was altogether absent and the right externus nearly so. So also a number of cases of paralysis of upward or downward movement are apparently due to symmetrical aplasia or mal-insertion of the superior recti and other muscles (Heuck, Lawford, Stieren, A. E. Davis). A similar paralysis of upward movement (Ormerod) or of both upward and downward movement (Uhthoff) may be caused by symmetrical lesions of the two third nerves at the base of the brain. In Thomsen's case of symmetrical paralysis of upward movement, the lesions were both basal and fascicular.

Conditions causing Conjugate Paralysis and Spasm.—Conjugate paralysis and spasm are often caused by *hemorrhage* (especially cerebral hemorrhage) and *embolism;* less frequently by *tumors, abscess* (especially otitic abscess, both cerebral and cerebellar), and *meningitis.* They may also occur in *polioencephalitis superior* before the stage of complete paralysis, indicating, then, that the process has involved the gray matter beneath the aqueduct, but not as yet the oculo-motor nucleus (Bernheimer).

Paralysis of conjugate movement, both lateral and vertical, occurs quite frequently in *multiple sclerosis* (in 3 per cent. of the cases, according to Uhthoff). It is also occasionally present in *pseudobulbar paralysis,* and, very rarely indeed, in *tabes* and *amyotrophic lateral sclerosis.*

Conjugate paralysis and especially conjugate spasm may be due to *hysteria,* the *traumatic neuroses,* or *neurasthenia.* In this case, Graefe's test (page 267) may show that the power of movement, although apparently absent, is actually present.

Defects in the associated movements of the eyes are sometimes *congenital* and *hereditary*. Such defects, however, are probably in most cases not instances of a true conjugate paralysis, but are simply the results of a symmetrical absence of the muscles or of some similar anomaly of development (page 272).

Treatment.—It is rare for a conjugate deviation to give rise of itself to any symptoms that require treatment. In cases in which the vertigo is annoying and the conjugate deviation persistent, the condition may be relieved by a symmetrical tenotomy (*e.g.*, of the two right rotators, if there is paralysis of levoversion). (A. Graefe.)

TREMOR OF THE ASSOCIATED PARALLEL MOVEMENTS.
NYSTAGMUS AND PSEUDONYSTAGMUS.

Varieties of Tremor.—We must distinguish three varieties of tremor of the eye movements:

A. Searching Movements.—Both eyes make an occasional wide, comparatively slow, sweeping movement from the primary position in some direction, and then, either immediately or after a time, return to the primary position. The movements are apparently purposeful, just as if the patient were trying to look at an object situated to one side of him. They may take place in any direction, vertical, oblique, or horizontal.

B. Pseudonystagmus.—One or both eyes when carried to a point near the limit of their excursion in some direction, make a series of jerky movements on from this point and back to it again, but in returning do not re-pass it. These jerks evidently represent an extreme effort to keep up the original excursive movement of the eyes, the smooth steady pull of the muscle that is carrying the eye along being converted into a series of discontinuous, spasmodic tugs.

The *direction* of the jerky movements in pseudonystagmus is usually the same as that in which the eyes were being carried when the movements started,—*i.e.*, if the eyes were moving to the right the pseudonystagmus will make them move jerkily still further to the right and then more slowly back again. Sometimes, however, when the eyes are moving laterally, the spasmodic attempt to carry them further may result, not in a continuation of the lateral movement, but in a jerky rolling of the eyes about an anteroposterior axis.

Pseudonystagmus, although more often *bilateral*, is not infrequently confined to one eye, particularly if that eye has relatively weak muscles.

C. Nystagmus.—In nystagmus the eyes make a series of very regular, short, quick oscillations about a central point. Nystagmus differs from pseudonystagmus, first, in that it often occurs when the eyes are in the primary position or near it, whereas pseudonystagmus occurs only when the eyes are near the end of an excursion in some direction; second, the movements in nystagmus are oscillations to and fro about a central point, while in pseudonystagmus they are movements from a terminal point and back to it again; third, the movements in pseudonystagmus are jerky and bespeak effort, in true nystagmus are smooth and regular, and, except in miners' nystagmus, are not often associated with a restriction in the excursive power of the eyes, which is very frequently present in pseudonystagmus.

Number and Extent of Oscillations.—The oscillations in nystagmus range from one to four a second and in width vary from one to five millimetres.

In nystagmus the oscillations may be *horizontal, vertical,* or *rotary,* —the cornea in the last case revolving like a wheel round its centre to and fro, through a more or less considerable arc. Combined vertical and horizontal nystagmus producing an oblique oscillation is called *mixed* nystagmus; and the same term may with propriety be applied to the forms in which a rotary is combined with a lateral or a vertical oscillation. Still *more complicated* are the cases described by A. Graefe as occurring in miners' nystagmus, in which, by a combination of successive horizontal and vertical movements, the centre of each cornea moves in a circular or elliptical path (*circumduction nystagmus*). Thompson and Spicer have described a similar circumduction movement in the nystagmus associated with spasmus nutans in infants.

Symmetry of Movements.—The nystagmic movements show a marked symmetry. The movements are nearly always bilateral, and when bilateral are almost, without exception, equal, simultaneous, and parallel in the two eyes,—*i.e.*, both eyes move equally far and at the same instant to the right, or both rotate to the right, etc.

Bilateral nystagmus is usually horizontal, although the rotary form is pretty frequent. Mixed nystagmus is less often met with, and vertical bilateral nystagmus is rare.

Very rarely indeed bilateral nystagmus is *disjunctive,*—*i.e.*, the two eyes, instead of moving in a parallel path, move towards or away from each other. Thus in Graefe's case of circumduction nystagmus the eyes both travelled in elliptical paths, but in opposite directions—each revolving from the nose towards the temple. I myself saw a case in which there was a converging rotary nystagmus,—*i.e.*, both corneæ made simultaneous wheel-like movements from the temple towards the nose. Thompson and Spicer say that infants affected with spasmus nutans sometimes show a form of nystagmus in which the eyes alternately and rhythmically converge and diverge, and the same form has been observed in adults as the result of excessive accommodation in near work (Eversbusch).

Also very rare are the cases of nystagmus in which the motions of the eyes are *dissociated,—i.e.*, are quite unlike, so that they do not resemble any co-ordinated movement, either parallel or disjunctive. Thus, the oscillations may be horizontal in one eye and vertical, rotary, or oblique in the other. Such combinations occur (*a*) in the nystagmus of spasmus nutans (Thompson); (*b*) sometimes in a unilateral nystagmus when it assumes the bilateral form (Schapringer, Neustätter, Duane); (*c*) occasionally in ordinary bilateral nystagmus (A. Graefe, Frost, Lawford, Duane).

Unilateral nystagmus is rare, some fifty-two cases having been reported. It is generally vertical, but was horizontal in cases described by v. Reuss, Everbusch, Bouchaud, Nagel, R. Sachs, Norrie (two cases), Neustätter, and Oppenheimer, and in one that I saw in Dr. Arnold Knapp's clinic. Unilateral oblique nystagmus has been seen by Neustätter, and unilateral rotary nystagmus by Weber, Simon (two cases), Neustätter, and myself. Simon saw one case in which for a time the nystagmus was unilateral and mixed (vertical and rotary), but later became bilateral and horizontal.

Sometimes when the nystagmus is apparently confined to one eye, it will be found that slight, inconstant oscillations are present in the other. These may be discovered only with the ophthalmoscope, which reveals a peculiar indistinctness of the fundus due to the tremor.

In other cases still, as proved by Simon's experience just cited, and in one case of mine, a unilateral nystagmus may become completely bilateral.

Finally, just the reverse may occur,—*i.e.*, a nystagmus originally bilateral may become confined to one eye. Thus, in spasmus nutans, the nystagmus may disappear from one eye before it disappears from both (Schapringer); and a bilateral nystagmus due to opacities of both corneæ may disappear first from the eye which clears the first (Simon).

Conditions causing Variations in Nystagmus.—Nystagmus is usually more or less intermittent. And, whether intermittent or not, nystagmus will in the same individual show considerable variations in both the amplitude and the rapidity of the oscillations. These variations are often determined by specific conditions,—*e.g.*, by physical and psychical excitation or depression, by sensory stimuli (particularly changes in illumination), by the use of alcohol, and sometimes even by voluntary effort (A. Graefe). Nystagmus is often seen to increase if the patient looks aimlessly into vacancy instead of fixing; or it increases if the eyes are carried in some special direction, ceasing perhaps if they are turned in another way. In other cases it ceases when the eyes are converged on a near object. Lastly, in some cases a nystagmus is regularly developed in one or both eyes as soon as one eye is occluded; in other cases such occlusion puts a stop to a nystagmus that is present in binocular vision.

Nystagmus may also change its direction,—*e.g.*, from vertical to oblique or even from vertical to horizontal, when the eyes are turned in different ways.

A careful study of the cases showing these variations will, I believe, lead to the conclusion that in a given case of nystagmus the oscillations become least in that position of the eyes which for this case involves the least strain upon the eye-muscles, or they are least under that condition of vision (exclusion of one eye, use of both eyes, use of glasses, etc.), which implies the least confusion to sight.

D. **Combined Forms of Tremor.**—In some cases we find searching movements combined with a pseudonystagmus. This would seem especially to occur when there is absence of central fixation, and at the same time the movements of the eyes are more or less restricted.

In some cases we may find nystagmus and pseudonystagmus combined in the same patient. Thus, as in a case of Uhthoff's, there may be a continual horizontal nystagmus which becomes coupled with a vertical jerky pseudonystagmus as soon as the eyes are carried far up or down.

Etiology of Nystagmus and Pseudonystagmus.—**Searching Movements** are found in blind eyes and in eyes which from chorioidal lesions at the yellow spot or similar causes have lost the power of central fixation.

Pseudonystagmus occurs almost constantly in hereditary ataxia, and in this disease it is usually very marked. It also is found very frequently in multiple sclerosis (in 46 per cent., according to Uhthoff). But as it is also present in a great variety of other nervous diseases, both functional and organic, and not infrequently, in fact, is found in people who are perfectly healthy, it is not of great importance as a diagnostic sign.

Pseudonystagmus in many cases, if not always, signifies some weakness of the muscles involved in making the special associated movement in the course of which it occurs.

Nystagmus is caused by—

(A) Conditions producing bilateral amblyopia in early infancy (*optical, infantile*, or so-called *congenital nystagmus*). Such conditions are opacities of the cornea, especially from blennorrhœa neonatorum; congenital cataract; hemorrhage, disease or abnormalities of the retina and chorioid; total congenital color-blindness, and albinism. Refractive errors, even when great, do not usually cause it, although they may do so.

Such nystagmus is probably not often really congenital, but develops during the first few months of life.

It must be remarked that unilateral amblyopia very rarely produces nystagmus but tends rather to produce squint. Simon, however, reports two cases in which an infantile unilateral nystagmus was caused by unilateral opacities of the cornea. It is noteworthy that in one of these cases the nystagmus was originally bilateral, and disappeared from one eye only when the cornea of that eye cleared up. Later, when the cornea of the other eye cleared, the nystagmus disappeared from that eye, too.

The unilateral nystagmus that occurs in an amblyopic eye affected with squint (Heimann) is probably not often infantile in origin, but is to be regarded as a late acquired optical nystagmus (see under type B, *infra*).

Sometimes a nystagmus of this form, evidently dating from infancy, occurs without there being any obvious lesion of the eyes to cause it. In such cases, according to A. Graefe, the nystagmus is probably attributable to a congenital retinal hemorrhage which subsequently cleared up.

(B) *Conditions developing in later life* causing constant strain of the eyesight from poor illumination or excessive strain of the eye-muscles, or usually strain of both kinds combined.

This variety of late acquired nystagmus is usually due to conditions incident to the patient's occupation (hence *occupational nystagmus*). The commonest form of this is *miners' nystagmus*, occurring in coal-miners who work with the eyes directed constantly up and back and by the insufficient light of the safety-lamps. The bad air of the mines is thought to be an additional factor in causing it. Miners' nystagmus is characterized by its markedly periodic character and its tendency to recur under conditions of reduced illumination, or when the eyes are directed up, or as a result of bodily exertion. Another feature, distinguishing it particularly from early acquired nystagmus (type A) is the fact that the eye movements are very greatly restricted and the patient finds it hard to keep the eyes in the fixing position (A. Graefe).

Similar to miners' nystagmus is the form occurring in compositors, paper-makers, metal-rollers, etc., who work with the eyes in a constrained position (Snell, Eversbusch), and in those who work in an insufficient light or under other conditions peculiarly trying to the eyes (Frost, Clarke). Magnus (cited by Snell) tells of the case of a baby who developed nystagmus as the result of constantly looking at a light above and behind his crib, and who got well when put in a properly lighted room.

In the same category belong those cases in which *optical nystagmus develops after the age of infancy* as a result of high astigmatism (as in a case of mine), or of traumatic cataract (Krauss), or of other conditions producing marked amblyopia. These cases are rare. They are comparatively often unilateral, and then are often, if not always, associated with squint, which may indeed be the cause of the amblyopia and the nystagmus (Heimann).

According to Bär a transient nystagmus may be induced by other forms of reflex sensory irritation originating in the eye,—for example, by inflammation or foreign bodies of the conjunctiva or cornea. Here the impulse for the discharge of the oscillatory movements presumably passes through the fifth nerve to the pons, finally reaching the abducens and oculo-motor nuclei. It is questionable whether in such cases the irritation of the eye is really the cause of the nystagmus, or whether it has not simply stirred into action a pre-existing nystagmus temporarily quiescent.

(C) Irritation arising from the ear (*aural nystagmus*). Nystagmus often is produced by some irritation arising from the inner ear. Such irritations appear to act by setting in motion the internus of the same side as the affected ear and the externus on the opposite side. It may be produced by actual disease of the labyrinth, by changes in labyrinthine pressure due to rapid rotation of the body, and by operative manipulations carried on in the middle and external ear (probing, syringing, etc.) (Eversbusch). Peters thinks that miners' nystagmus is of labyrinthine origin.

According to Schwartze, aural nystagmus is not due primarily to heightened labyrinthine pressure, but to irritation of the vestibular nerve.

(D) *Nervous Disease.*—True nystagmus occurs frequently in multiple sclerosis (in 12 per cent. of the cases, according to Uhthoff). It is also frequent in syringomyelia.

Nystagmus has also been described as occurring often in other nervous diseases, as meningitis, meningeal hemorrhage, sinus-thrombosis, hemorrhage and softening of the brain, tumors of the brain, particularly the cerebellum, and pons disease (Gowers). In many of the reported cases, however, care has not been taken to distinguish between nystagmus and pseudonystagmus. Except in multiple sclerosis and syringomelia, and in conditions due to ear trouble, true nystagmus is rare in nervous disorders; occurring in only 3 cases out of 500 examined by Uhthoff and in only 1 per cent. of tabics. And even in the cases in which it does occur it is often not due to the nervous disease itself, but is simply an infantile nystagmus produced by opacities in the media, congenital defects, etc.

We may, therefore, say that a true nystagmus, not due to optical defect nor dating from childhood, and not traceable to occupation, as in miners' nystagmus, nor to ear disease, is strong presumptive evidence *either of multiple sclerosis or less probably of syringomyelia.*

A transient nystagmus, often of peculiar form, frequently occurs in *spasmus nutans* (Thompson, Spicer, Schapringer). This may be unilateral, or be bilateral at one time and unilateral at another.

Nystagmus may occur in *hysteria;* and in hysterical subjects has been evoked and cured by suggestion (Sabrazès).

Nystagmus, probably due to central nervous disease, has been occasioned by *traumatism.* Such nystagmus is rare. It may be transitory, as in two cases described by Norrie. In some instances, at all events, as in those given by Apelt, it seems to be simply the result of a functional nervous condition (traumatic hysteria).

Not to be confounded with these cases of traumatic nystagmus are those in which an acquired optical nystagmus (see under type B) has resulted from the optical defects produced by a traumatism (two cases described by Krauss).

Both nystagmus and pseudonystagmus occurring in nerve disease are often associated with paresis or weakness of the muscles.

(E) *Poisons.*—Nystagmus is said to occur very often in patients that are under the influence of ether. Other agents that very rarely produce nystagmus are alcohol, cocaine, sulphonal, arsenic, lead, quinine, ergot, and sewer gas (Uhthoff).

(F) *Voluntary Nystagmus.*—Occasionally nystagmus may be produced voluntarily (A. Graefe; Dodd; A. E. Davis). Graefe thinks that this can occur only in those who were formerly the subjects of an involuntary nystagmus, which has disappeared.

Nystagmus, whether infantile or due to nervous disease, is sometimes *hereditary* (Nettleship; McGillivray; Audeoud; see also excellent summary by Groenouw).

Site of Lesion in Conditions of Ocular Tremor.—In the *infantile* form of **nystagmus**, as well as in the form due to occupation (*miners' nystagmus*), and to *aural irritation*, the tremor is not due to any actual lesion of the brain. The contrary has been asserted of miners' nystagmus, but, as A. Graefe says, without sufficient proof. In the nystagmus of multiple sclerosis and other *organic nervous diseases*, lesions have been found mainly in the corpora quadrigemina, corpus striatum, restiform bodies, cerebellum, and medulla. It may be noted that experimental stimulation of the same parts and also of the cerebral cortex in animals likewise produces nystagmus. Occasionally the nystagmus in nervous disease seems to be produced not by a lesion interfering directly with the paths for the associated movements of the eyes, but by remote lesions which act by causing variations in the intracranial pressure (Uhthoff). (See Pages 449 and 472).

In **pseudonystagmus** due to organic nerve disease the lesions are also in most cases central, although in some instances they may be seated in the peripheral nerves supplying the muscles.

Theory of Nystagmus.—No perfectly satisfactory explanation of nystagmus has yet been given. The remarkable regularity and parallelism of the movements, however, show that in nystagmus we have to deal with a *perversion of the centres for parallel and parallel-rotary movements*, and not with peripheral lesions of the muscles themselves or their nerves.

This perverted state of the association centres is evidently produced in optical and miners' nystagmus by long-continued abnormal stimuli passing up through the visual tract, and in miners' nystagmus also through the fifth nerve; in aural nystagmus by abnormal reflexes passing through the auditory nerve to the centres in the pons and perhaps also in the corpora quadrigemina; while in nystagmus due to nervous disease, the association centres and tracts are probably affected directly.

Nystagmus in all probability is produced by alternate, just as normal fixation is produced by simultaneous, discharges of motor energy from the two sides of the brain. That is, when we fix, the eyes are held steadily in place, because impulses are sent down at the same time from the right angular gyrus (which acting alone would cause both eyes to move to the left) and from the left angular gyrus (which acting alone would cause both eyes to move to the right). In a person with nystagmus the impulses from the right angular gyrus and from the left angular gyrus follow each other in rapid succession.

Nystagmus, then, may be regarded as a kind of *imperfect or perverted fixation.* Infantile nystagmus, in particular, is an incompletely developed fixation—a condition intermediate between the wide, more or less irregular, excursions of a congenitally blind eye and the steady gaze of an eye which fixes normally.

This explanation of the nature of nystagmus holds good undoubtedly even for the rare *unilateral and dissociated* forms. These differ from the ordinary bilateral and symmetrical nystagmus only in the fact that either the reflex discharges from the association centres, which cause the oscillation, are in some way so altered as to affect one eye more than the other, or else there is a contributing cause,—usually some muscular anomaly,—which being mainly seated in one eye makes that eye respond more readily than its fellow to the abnormal stimuli.

Symptoms of Nystagmus.—The symptoms of nystagmus are—

1. *Apparent Movement of Objects.*—We should expect that the rapid oscillation of the eyes in nystagmus would make it seem to the patient as if all objects that he looked at were jumping to and fro. And in miners' nystagmus and the form due to aural disease this apparent movement of objects is a very prominent and distressing symptom. It is also often, though not always, present in the nystagmus due to nervous disease. Sometimes in these cases the patient not only sees objects move but feels that the eye is trembling.

On the other hand, in optical nystagmus due to infantile amblyopia, this apparent movement of objects is very rarely perceived. Sometimes in unilateral nystagmus and, under exceptional conditions, in bilateral nystagmus it can be evoked (A. Graefe). Even then, however, it seems hardly ever to be troublesome.

2. *Reading in Vertical Lines.*—That the apparent movement of objects, even when it is not perceived by the patient, yet causes indistinctness of things seen, is shown by the fact that some patients with horizontal nystagmus in reading hold the book sideways, so that the lines of print run vertically. This, to a certain extent, obviates the confusion produced by the oscillations of the letters (Soelberg-Wells, Wecker, Duane).

3. *Vertigo.*—This is marked in some of the late acquired forms, especially in miners' nystagmus and the form due to aural irritation.

4. *Nodding Movements of Head.*—These often occur in optical nystagmus. They were formerly thought to be compensatory in character, acting to prevent the tremor of objects produced by the oscillation of the eyes. This, however, is certainly not the case; indeed, in one instance

that I saw the nodding of the head considerably increased the confusion of sight due to the nystagmus.

5. *Poor Vision.*—In most cases of nystagmus of infantile origin, the vision is quite poor, and cannot be brought up to normal by glasses. This is doubtless in part due to the blurring that the nystagmus produces, but much more to the pathological condition causing the nystagmus. In fact, nystagmus in itself does not necessarily cause permanent poor sight. I have seen it associated with a vision of nearly $\frac{20}{20}$. It is to be inferred that in such cases the application of glasses or the mere act of fixing attentively with the moving eye has made the latter quiet for the time being, since it is very difficult to suppose that the sight could be anything but bad so long as the eyes are still in active motion. In fact, we can prove in those cases in which the nystagmus increases greatly in one direction of the gaze and diminishes in another, that the sight is very much better in that position in which the oscillation is slight. In miners' nystagmus the disturbance of sight produced by the tremor is considerable.

6. *Photophobia* may be present. In unilateral nystagmus it may occur only when the oscillating eye is used to see with (Schapringer).

Course of Nystagmus.—Infantile nystagmus generally lasts through life, although it may disappear. Nystagmus due to aural or nervous disease naturally persists as long as its cause. Miners' nystagmus disappears if the patient gives up his work and lives under proper conditions as regards illumination and eye-strain. Nystagmus due to spasmus nutans and hysteria is probably always transient. A transient nystagmus may also result from traumatism (Norrie), tenotomy (Wecker), or other causes (Simon, and others).

Treatment.—The treatment of nystagmus must be directed to the cause. Infantile nystagmus is rarely affected by any treatment, although isolated cases have been cured by removal of a congenital cataract (Crzellitzer, Cuperus), by tenotomy or advancement causing relief of a squint (Kugel, Neustätter, Heimann, Cuperus), by exercises with a stereoscope and in reading with the aid of a perforated diaphragm (Lavagna), by exercises in fixation in various positions of the gaze (Brisscaud; Meige and Feindel), and by exercises with rotating prisms (Trombetta). As a temporary help Kugel recommends protracted bandaging of the eyes and the use of dark glasses before the better eye. Some cases of nystagmus, especially acquired nystagmus,
lieved by the correction of astigmatism or other refractive errors
Duane). In miners' and other forms of occupational nystag-
ge of occupation is imperative.

SECTION VI.

ANOMALOUS DEVIATIONS OF THE EYES.

DISJUNCTIVE MOVEMENTS.

In some cases the eyes no longer follow the law of associated movements, but each moves irrespective of the other. This occurs, for example, in eyes that have been totally blind from birth, also in patients deeply under the influence of chloroform. But it occasionally occurs in other cases, too.

Thus, a *deviation of one eye down and out and of the other eye up and in* is said by Gowers to occur in disease of the middle cerebellar peduncle.

Lawford describes a case in which, when the patient was told to look to the left, *both eyes moved outwards.* (This may have been simply an instance of excessive voluntary divergence of the eyes—divergence-excess).

Kunn gives six cases of *dissociated paralyses.*

Uhthoff says that occasionally *dissociated ataxic movements* occur in tabes.

I have seen a case in which the right eye at intervals moved slowly up and after awhile moved down again, while the left eye remained perfectly still. The movement of the right eye took place whether the left eye was covered or open. The condition was probably a sort of *unilateral searching movement* (page 273), and may be regarded as an abortive form of unilateral nystagmus.

A special form of partial dissociation of the ocular movements is seen in those cases in which on alternate covering *each eye goes up* or *each eye goes down* behind the screen. Cases of this sort have been described by Schweigger and especially by Stevens. I myself have seen three or four. In these cases there may or may not be binocular fixation when both eyes are uncovered. If there is binocular fixation, the condition is called *anophoria* or *double hyperphoria* if both eyes go up, and *catophoria* or *double hypophoria* if both go down, when screened. The corresponding conditions in which one eye squints when both are uncovered are called *anotropia* and *catotropia.* Such a squint may be alternating, and then in anotropia each eye in turn will squint up (*double hypertropia*) and in catotropia each eye in turn will squint down (*double hypotropia*).

Some of the cases described as illustrating dissociation of the eye movements *are not really of this character.* Thus, Konigshöfer's case in which, without moving the right eye, the left could be voluntarily turned in or out or be made to look straight ahead, was evidently one of left divergent squint, which was overcome by a strong effort of convergence, both eyes at the same time being carried to the right by an associated parallel movement. Again, the case given by A. Graefe in which the left eye went straight up when the attempt was made to move both eyes to the right, can readily be explained by supposing that, as the left eye could not move inwards because its internus was paralyzed, it was forced spasmodically upward by the overaction of its elevators, which were brought excessively into play. (*Cf.* remarks, page 249.)

Weinhold's case, in which a patient with usually normal eye movements could at will produce a left strabismus sursumvergens, marked when he looked to the right but more and more slight when he looked to the left, was probably one of moderate insufficiency of the right superior rectus. In this case, if the patient fixed with the left eye the deviation would be but slight, even when he looked to the right, and might indeed be absent altogether if, as could well happen, he had developed an excessive ability to diverge the eyes in a vertical direction. If, however, by a voluntary transition he fixed with the paretic right eye, the left eye would immediately assume a position of marked secondary deviation and would shoot up, especially when he looked to the right.

An apparent dissociation of the ocular movements, furthermore, may exist in those cases in which several muscles are paralyzed in one eye and yet this eye is used for fixation. Here, as A. Graefe points out, the secondary deviations of the sound eye, rapidly changing their form and varying their amount as the eyes are carried in different directions, make it appear as if the eyes were moving quite contrary to the ordinary laws of association.

CHAPTER VI.

THE INTRA-OCULAR MUSCLES.

By JOHN E. WEEKS, M.D.

IRIS.

The iris is the terminal portion of the vascular tunic. Its free border, which describes a circle enclosing the space known as the pupil, rests on the anterior surface of the capsule of the lens. As the lens surface occupies a plane anterior to the ciliary border of the iris, the iris forms a shallow cone, the depth of which depends on the position of the crystalline lens. If the lens is absent, the iris hangs in a vertical plane and is tremulous.

The diameter of the iris (in small eyes 9 millimetres, in large eyes 13 millimetres) is approximately 11 millimetres. The iris is not of uniform thickness, but averages 0.04 millimetre when at rest. When widely dilated it may double in thickness.

At the insertion of the iris the posterior two-thirds pass into the ciliary body. The anterior third is continuous with the ligamentum pectinatum (Compare Page 22).

Anterior Endothelial Layer.—The iris is limited anteriorly by a layer of flat, nucleated endothelial cells, which is continuous over the surface of the trabeculæ of the ligamentum pectinatum and the posterior surface of the cornea.

Vascular Stroma Layer.—The " vascular stroma layer" is composed of bundles of connective tissue loosely associated. This layer contains numerous blood-vessels, nerve-trunks, and irregular lymph-spaces. Round, oval, and branching nucleated cells, the protoplasm of which becomes pigmented in colored irides, are found in this layer, as are also the muscles of the iris—the sphincter and dilatator pupillæ.

The **sphincter pupillæ** muscle lies very near the pupillary margin. It is a flat, circular band, measuring about one millimetre in width, varying slightly according to the state of contraction, and is about one-tenth as much in thickness. It is composed of bundles of non-striated muscle-fibres which interlace. The sphincter pupillæ is separated from the pupillary margin by the posterior pigment layer of the iris, which extends a very little way over the stroma at the pupillary margin and is seen as a narrow, black ring at this point.

The **dilatator pupillæ** consists of two or more layers of long spindle-shaped cells possessing elongated nuclei. They are undoubtedly non-striated muscle-fibres. These fibres are arranged meridionally and lie on the limiting membrane. They apparently form a continuous layer extending from the ciliary margin of the iris to the sphincter pupillæ, with which they blend, entering at the outer border and posterior surface.

Lamina Vitrea, Membrane of Bruch.—This extremely delicate membrane is homogeneous under low magnifying powers, but presents fine striations when examined with high powers of the microscope. It is extremely thin (0.002 millimetre) and is closely adherent to the inner layer of cells of the pigment layer.

Pigmented Layer (Uvea of the Iris).—This represents the extreme anterior portion of the secondary eye vesicle. The cells of the deeper layer are somewhat flattened, are spindle-shaped, and are arranged radially to the pupillary margin. They rest upon the lamina vitrea of the iris. The cells of the outer layer are short, cylindrical or polygonal cells. It is now held by some histologists that the inner layer of cells which forms the pars iridis retinæ is concerned in the development of the sphincter and dilator muscles of the iris. At the ciliary attachment, the pigment layer of the iris is continuous with the pigmented layer of the ciliary body, the pars ciliaris retinæ.

Vessels of the Iris.—The arterial supply to the iris is derived from the long posterior ciliary arteries and from the anterior ciliary arteries. The capillaries of the iris terminate in the venæ radicæ which proceed radially to the ciliary body; they empty into the venæ vorticosæ. The walls of the arteries are thick, due to a preponderance of connective tissue without elastic fibres. Muscle-fibres are said to be relatively few. The arteries and veins possess thick sheaths of connective tissue, between which and the vessel wall proper the perivascular lymph-space is found.

Lymphatics of the Iris.—There are no distinct lymph-vessels in the iris. The clefts between the tissue elements of the anterior border layer, the irregular spaces between the bundles of fibres of the vascular stroma and the perivascular spaces, are the lymph-channels of the iris. They communicate with the lymph-spaces in the ciliary body, with the lymph-spaces at the filtration angle, and through the spaces of Fontana with the anterior chamber.

Nerves of the Iris.—The bundles of nerve-fibres that enter the iris are derived from the ciliary plexus that lies in the ciliary muscle. Three kinds of fibres are given off from this plexus: (1) The sensory; (2) the motor; (3) the sympathetic. The motor-fibres from the third nerve pass to the sphincter pupillæ. The sympathetic nerve-fibres pass backward toward the dilatator and are supposed to supply it (Baker).

Function of the Iris.—The function of the iris is to regulate the quantity of light that enters the eye so that the act of vision will be carried on with the greatest possible efficiency. The iris is a diaphragm which changes the size of its central opening, the pupil, as the conditions demand. The movements of the iris are involuntary and are made unconsciously. They are induced by stimuli which act through certain "reflex arcs." The stimuli which produce these movements of the iris are light, cutaneous sensations, emotions, convergence, accommodation, association with certain movements of the eye and eyelids. Stimulation of the motor oculi nerves causes contraction of the sphincter iridis with consequent narrowing of the pupil. Paralysis of this nerve causes the sphincter to relax, dilating the pupil. Stimulation of the ciliospinal centre in the spinal cord, or of the superior portion of the cervical sympathetic, causes contraction of the dilator of the iris, resulting in a widening of the pupil to a greater extent than obtains in paralysis of the motor oculi. Paralysis of the sympathetic causes a narrowing of the pupil (miosis) greater than that which obtains when the third nerve is also paralyzed.

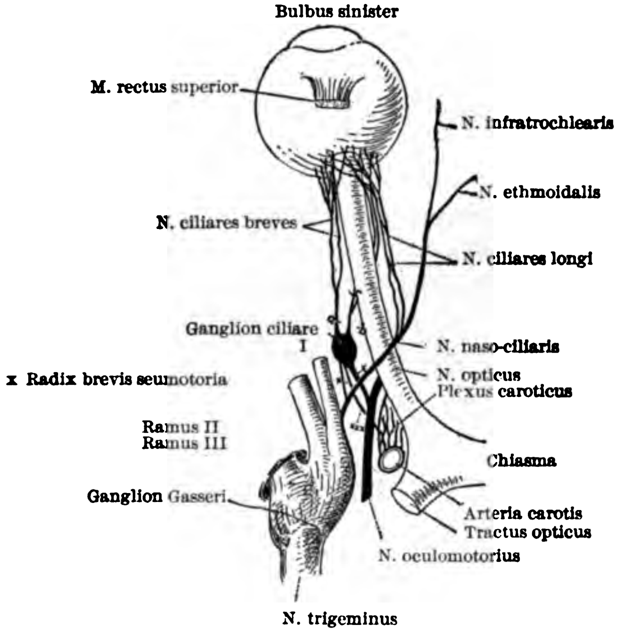

FIG. 1 illustrates the anatomical relation of the short and long ciliary nerves: x, short root of the ciliary ganglion; motor (from the oculomotor nerve) xx, long root of the ciliary ganglion; sensory (from the trigeminus) xxx, sympathetic root of the ciliary ganglion (from the carotid plexus of the sympathetic nerve). (Piltz).

Normal Pupil.—The size of the pupil varies greatly in different individuals and in different degrees of illumination, ranging from 1.5 millimetres by bright illumination to 9 millimetres by weak illumination. The average diameter in young adult life by moderate illumination is 4 millimetres. In infants the pupil is small; in youth, large; and in the aged it is again small. Lange[1] measured 1000 pupils in patients of different ages and sex. His conclusions were as follows: (1) The size of the pupil differs greatly in different persons. (2) As a rule, women have larger pupils than men. (3) The size of the pupil decreases with age. (4) The width of the pupil is less in hypermetropic eyes than in emmetropes and greater in myopes than in emmetropes. (5) After the fortieth year the difference in the width of the pupil in different states of refraction is inconsiderable.

The position of the pupil is, as a rule, a little to the nasal side of the centre of the iris. The pupils change in size symmetrically. Marked inequality in the size of the pupils is usually a pathological phenomenon. Slight differences in the size of the pupils frequently exist under normal conditions. Iwanow[2] examined 134 military recruits, finding an equal width of the pupils in only twelve; the right pupil was larger in 49; the left in 73. Felton[3] found an inequality of the pupils in 28 of 61 persons examined.

EXAMINATION OF THE IRIS.

In the examination of the iris a number of methods are employed.

1. **Simple Inspection.**—For this, moderate illumination is necessary. When the pupils are compared, the illumination of each eye should be equal.

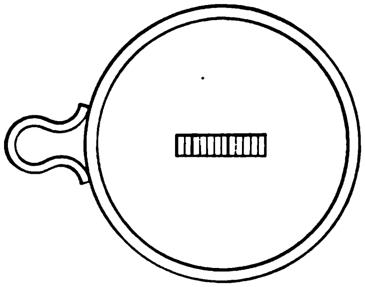

FIG. 2.—Priestley-Smith keratometer.

2. **Oblique Illumination.**—This is of service for the purpose of throw-

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[1] Inaug. Dissert., Amsterdam, 1901.

[2] Vratsch, No. 7, 1887.

[3] Inaug. Dissert., Bonn, 1895.

ing light on various parts of the retina in studying the pupillary light reflex.

3. **Lenses.**—These aid in determining minute movements of the iris. They may be simple or compound. Compound lenses are monocular or binocular. A monocular instrument that has given some degree of satisfaction is that devised by Howe of Buffalo and manufactured by Meyrowitz of New York. The monocular instrument is inferior to the binocular, since with the binocular compound lenses stereoscopic vision is obtained. Jackson's binocular loupe adjusted to a head-band is a very good instrument. Another, and probably the best, is the corneal microscope of Czapski, made by Zeiss of Jena.

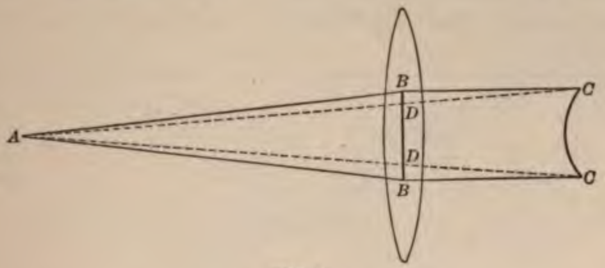

FIG. 3.

Methods of Measuring the Iris.—Measurement of the diameter or width of the iris may be accomplished by means of a small millimetre measure, placing the measure before the eyes as close to the cornea as possible. The width of the iris at its line of insertion at the ciliary body is somewhat hidden by the anterior border of the sclera at the sclero-corneal margin. This fact must be taken into consideration in making the measurements just referred to. The keratometer of Priestley-Smith (Figs. 2 and 3) may also be employed for this purpose.

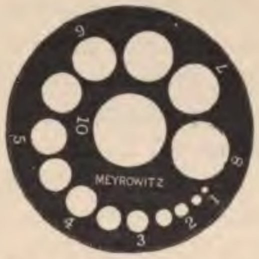

FIG. 4.—Circular pupillometer.

The size of the *pupil* may be measured by means of a millimetre rule, but, if this is employed, it must be remembered that the size of the pupil is actually greater than it appears to be on the rule and that the difference in size is in direct proportion to the distance of the rule from the pupil, since the observer's line of vision cuts the rule before reaching the pupil. A very simple and excellent method is by means of the ordinary circular pupillometer (Fig. 4). The little disk is held close to the cornea and is rotated until an aperture in the margin of the disk corresponds to the size of the pupil. The diameter of this opening, which is marked on the disk, is then recorded.

There are many other pupillometers that may be employed. The pupillometer of Soleberg Wells, of Hirschberg (Fig. 5), of Landolt,[4] of Schirmer,[5] etc., but these possess little advantage over the simple one first mentioned.

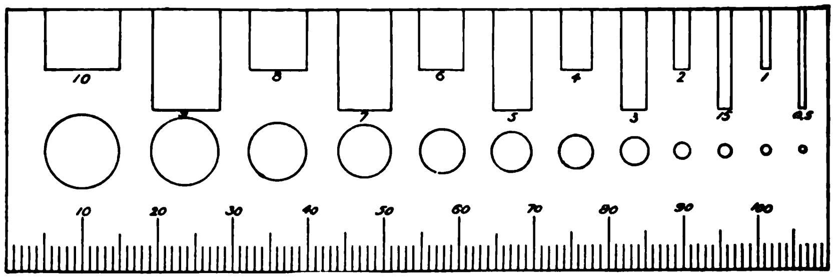

FIG. 5.—Hirschberg pupillometer.

LIGHT REFLEXES.

The reaction of the pupil to light is excited (1) by the direct entrance of light, known as the *direct* light reflex; (2) by the entrance of light into one eye, the observed eye being shaded—the *indirect* or *consensual* light reflex. Both reflexes are due to the integrity of what is termed the *light reflex arc,* which consists of fibres running from the eye to the brain, known as centripetal fibres, and fibres running from the brain (nucleus of the third nerve) to the iris, centrifugal fibres.

The centripetal fibres have their origin in the retina (according to Coppez[6] in the amacrine cells, but this is not fully proven), being apparently most plentiful in the region of the posterior pole, diminishing in number towards the periphery, apparently having the same distribution as the fibres concerned in vision, that is, those that decussate come from the nasal half of each retina, the non-decussating from the temporal half. They pass into the optic nerve and, according to von Gudden, von Monakow, Bernheimer,[7] and others, can be differentiated

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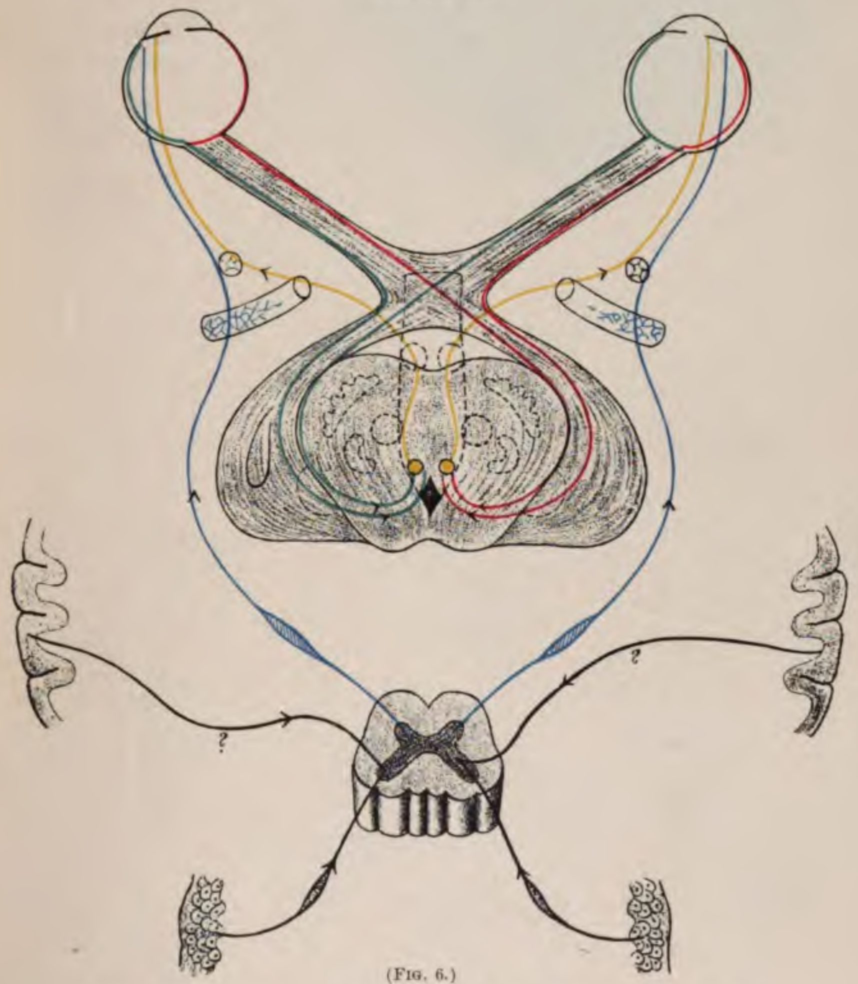
[4] Landolt. De Wecker et Landolt, Traité complet d'Ophthalmologie, p. 942.

[5] Schirmer, Archiv f. Ophth., xl. 5.

[6] Coppez. Revue générale. February 28, 1903.

[7] Bernheimer, Archiv f. Ophth., Bd. xlvii. S. 1.

PLATE II.



(FIG. 6.)

Pupillary Light Reflex Arc; Sympathetic, Cortical, and Sensory Dilator Tracts.

Red and green.—Centripetal tract of the light reflex arc (crossing at the sphincter centres not shown.)

Yellow.—Centrifugal tract of the light reflex arc.

Blue.—Sympathetic tract containing the nerve-fibres which innervate the dilator pupillæ.

Black.—Cortical and sensory tracts. (Uhthoff.)

from the fibres that have to do with vision by their larger size. Passing into the chiasm, they there undergo semi-decussation and proceed along the optic tracts to the outer geniculate bodies, where, according to Bernheimer, they bend inward, pass to the anterior part of the anterior corpora quadrigemina, and thence to the sphincter nucleus in the anterior portion of the nucleus of the third. Bernheimer is not positive that the centripetal fibres pass into, but thinks that they may end in arborations at the sphincter nucleus. Experimental and pathological data at hand make it probable that the centripetal fibres of the light reflex arc do not pass through the anterior corpora quadrigemina. Moeli[8] reports a case of absence of light reflex in which the lesion was found to be a tumor involving both sides of the posterior walls of the third ventricle, making it probable that the fibres referred to pass in these walls. In this case the pupils were 5 millimetres in diameter, equal in size; vision, motility of eyes, and convergence (page 301) reflex normal.

The experiments of Ferrier and Turner[9] seem to prove conclusively that the fibres do not pass into the corpora quadrigemina. Young monkeys were employed. The corpora quadrigemina were exposed by opening the skull and removing the occipital lobe of the brain backward from the parieto-occipital fissure. The anterior quadrigeminal bodies were then destroyed with the cautery. This did not abolish the pupillary light reflex. The movements of the iris were affected only when the lesion extended into the underlying Sylvian gray matter. Clinical evidence to this effect[10] is also obtainable.

Bach[11] is of the opinion that the centripetal bundle of fibres *divides* at or near the external geniculate body one part proceeding to a centre in the lower part of the medulla, which he terms the inhibitory centre, fibres returning from this centre to the sphincter nucleus. This supposition is not proven and in the light of the experiments of Ruge,[12] who divided the medulla some distance above its lower extremity without abolishing or changing the pupillary light reflex, it is highly probable that this centre does not exist.

In regard to the effect of lesions of this tract, the experiments of von Bechterew[13] on dogs led to the following conclusions:

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[8] Moeli, Archiv f. Psychiatrie, Bd. xviii. S. 1.

[9] Ferrier and Turner, Brain, 1901, vol. xxiv. p. 27.

[10] Goldzieher and Bechterew, Archiv f. w. ges. Physiol., Bd. xxxi., S. 60.

[11] Bach, Graefe's Archiv, Bd. lvii., H. 2, p. 239.

[12] Ruge, Graefe's Archiv, 1902, Bd. liv., H. 3.

[13] Von Bechterew, Archiv f. w. ges. Physiol., Bd. xxxi., S. 60.

1. Monolateral lesion of the sphincter nucleus causes loss of the light reflex of the eye on the same side and loss of the consensual reaction in the opposite eye.

2. A lesion of one side of the third ventricle induces one-sided loss of light reflex with good vision.

3. A lesion affecting both sides of the wall of the third ventricle causes loss of light reflex in both eyes, with normal sight.

Decussation of the Fibres of the Pupillary Light Reflex Arc.—The question of semi-decussation in the chiasm is apparently settled. Although there is as yet no positive clinical proof, the experiments of Bernheimer are conclusive. Bernheimer divided the tractus of one side in monkeys and then found that the pupillary light reflex, both direct and consensual, was preserved. Decussation or intercommunication at the sphincter nuclei is evident both from clinical and from experimental observation. The case of S. Weir Mitchell[14] furnishes almost, if not quite, conclusive clinical evidence. The chiasm was cut in half sagittally by a tumor which divided all but a very few fibres. There was bitemporal hemianopsia with retention of the pupillary light reflex. Bernheimer divided the chiasm in monkeys by sagittal incision and demonstrated the retention of the pupillary light reflex. Whether the pupillary light reflex was preserved in the nasal portions of the retinæ is not stated. Theoretically, this should not occur.

Lesions Affecting the Pupillary Light Reflex Arc; Centripetal Fibres.

1. *Lesions affecting the retina or optic nerve* on one side producing blindness cause loss of direct pupillary light reflex on the affected side and consensual light reflex on the opposite side. The consensual reaction is preserved on the blind side. With uniform illumination the pupils are of the same size, as a rule; in some cases the pupil of the blind eye is a trifle larger than its fellow in moderate and bright illumination when both eyes are equally exposed. If, by moderate illumination, the seeing eye is covered the pupil of the blind eye dilates. If there is amblyopia (blindness not complete) the direct pupillary light reflex in the affected eye and the consensual pupillary light reflex of the fellow-eye are not entirely abolished, but are less active than in the normal eye.

2. *Sagittal section of the chiasm* will, since the distribution of the fibres of the pupillary light reflex in the retina correspond to the distribution of the fibres concerned in vision, cause loss of direct and consensual pupillary light reflex in each eye when the nasal half of the retina

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[14] Jour. of Nerv. and Ment. Dis., January, 1889.

only is illuminated.[15] Illumination of the temporal halves of the retinæ elicit both direct and consensual pupillary light reflex. With this lesion the pupils are slightly larger than normal.

3. *Sagittal section of the chiasm and lesion of one optic nerve*, with consequent blindness on one side and temporal hemianopsia on the other side, cause complete loss of direct pupillary light reflex in the blind eye and of consensual pupillary light reflex in the seeing eye; complete loss of direct pupillary light reflex in the seeing eye and loss of consensual pupillary light reflex in the blind eye when the *nasal half* of the seeing eye is illuminated, and retention of the direct pupillary light reflex in the hemianopic eye and of consensual pupillary light reflex in the blind eye when the *temporal half* of the retina is illuminated. The pupil of the blind eye will be slightly larger than that of the fellow-eye by bright and moderate illumination, but of the same size in the dark (as may be demonstrated by flash-light photography).

4. *Lesion of the tractus* on the right side produces left homonymous hemianopsia, with loss of the direct pupillary light reflex in the illuminated eye and loss of the consensual pupillary light reflex in the fellow-eye (absence of the Wernicke pupillary light reflex) when the right half of the retina of either eye is illuminated. Illumination of the left, or seeing half, of either retina elicits direct pupillary light reflex of the illuminated eye and consensual pupillary light reflex of the fellow-eye. The pupils in this condition are slightly larger than normal (other things being equal) by moderate illumination.

5. *Lesion of a tractus* and destruction of the same half of the chiasm or of the optic nerve on that side produce the same clinical conditions as those described in 3.

6. *The lesions* heretofore described are accompanied by blindness of the corresponding parts of the retinæ. If it were possible to destroy by lesion the bundle of fibres of the pupillary light reflex arc between an optic tractus and the sphincter nucleus, there would be no blindness, but there would be loss of pupillary light reflex corresponding to that produced by lesion of one tractus if one side only was involved and complete loss of direct and consensual pupillary light reflex on both sides if the bundles on both sides were destroyed. The pupils would remain of normal size. Lesions at this point do not abolish the convergence light reflex.

Sphincter Nuclei.—The sphincter nuclei, according to Bernheimer,

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[15] In testing the reflex with such a lesion, the difficulty in excluding light (see page 302.) from the opposite part of the retina must be borne in mind.

Parlia, Kahler and Pick, and others, lie in the anterior part of the nucleus for the third, at the posterior boundary of the third ventricle, separated from the nucleus for the ciliary muscle. Bernheimer describes a central communication of the two sphincter nuclei, and is of the opinion that the communication is by means of processes from the neurons of either nucleus.

Centrifugal Fibres of the Pupillary Light Reflex Arc.—These fibres arise from the sphincter nucleus of the side corresponding to the iris which they are to innervate, pass into the underlying tissue of the pons, where they are joined by fibres from the ciliary nucleus. They then pass into the trunk of the third nerve occupying, according to Ackerman, the central portion of this nerve-trunk; then by the short root to the ciliary ganglion; then by the short ciliary nerves (five to seven in number) to the posterior part of the globe, passing through the sclera, and, forward between choroid and sclera, into the ciliary body, from the anterior portion of which they pass into the iris tissue and to the sphincter.

Lesions of the Centrifugal Fibres.—Since the centrifugal fibres after leaving the sphincter nuclei do not decussate, and since they are joined by the fibres from the ciliary nucleus soon after their origin and are incorporated with the trunk of the third nerve before it leaves the pons, it is possible to have an isolated lesion of the pupillary light reflex arc only at or very near the sphincter nucleus. Caspar [16] reports a case as follows: P., aged 38 years; loss of direct and consensual pupillary reflex with preservation of vision; pupil responds to convergence. He places the lesion between the sphincter nucleus and the point where the fibres from the ciliary nucleus join the pupillary light reflex bundle. Wilder [17] reports two cases of monocular iridoplegia, in both of which there was paresis of accommodation. There was complete preservation of convergence pupillary reflex. Such a lesion produces loss of pupillary light reflex direct and consensual on the side affected. It produces paralysis of the sphincter muscle, but does not affect direct or consensual reaction in the fellow-eye. Affections of this bundle in other parts of its course are almost invariably associated with other third-nerve paralyses. If the centrifugal pupillary light reflex bundle on one side only is affected, the pupil on the corresponding side is larger than its fellow, except in the dark, when (flash-light photography) they are of the same size. If the bundle on each side is involved, both pupils are dilated in

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hiv f. Augenheilk., Bd. xxvii. p. 291.

. of Ophthal., April, 1897.

all degrees of illumination. They should be equally so, but Heddaeus states that anisocoria sometimes exists.

Complete section of the third nerve produces total loss of the pupillary light reflex and consensual light reflex on the affected side.

Convergence Pupillary Reflex.—When, under normal conditions, the eyes change their regard from a distant to a near object, or, in other words, when the visual lines are simultaneously deflected from parallelism to extreme convergence, the pupils contract very markedly. This phenomenon is called the *convergence pupillary reflex.* It is most pronounced in eyes whose irides are most active and in those which, with normal irides, have large pupils, as in children and young adults. In the aged, whose pupils are small, particularly in those whose cervical sympathetic nerves are paralyzed, and in tabetic patients, as well as in all who possess the Argyll-Robertson pupil, the convergence pupillary reflex causes but slight movement of the iris.

This pupillary reflex is often described as a convergence and accommodation reflex and sometimes as an accommodation reflex. It is very easy to demonstrate the fact that the reflex is *much* more closely related to convergence than to accommodation. In many cases it is impossible to determine that accommodation influences the pupil at all. The difficulty in eliminating convergence from accommodation makes it almost impossible to determine just what influence accommodation has in the production of this reflex, but it is certain that some pupillary reaction accompanies accommodation in many individuals.

The *amplitude* of the contraction depends on the size of the pupil and on the activity of the iris.

In the emmetropic and hypermetropic eye, the amplitude and promptness of the reaction is greater than in myopes.

In pupils of unequal size, the amplitude of movement is greater in the eye with the larger pupil, other conditions being equal.

If one eye is amaurotic, the fellow-eye being good, the reflex is the same. Contraction of the pupil also occurs on convergence if both eyes are amaurotic, possibly not to so great a degree as in seeing eyes.

If there is no reaction of the pupil in convergence, paralysis of the sphincter is to be suspected.

This pupillary reflex disappears, as a rule, with absence of convergence, whether the absence is voluntary, that is, is not exercised, as in cases of loss of one eye, in which case the act of convergence is often abandoned after the lapse of some time, or in true absence or paralysis of convergence.

The frequent association of loss of accommodation, paralysis of the ciliary muscle and of the sphincter pupillæ, with loss of convergence, points to the close association of the sphincter and ciliary centres in the nucleus of the third with the portion of the brain controlling convergence, but no anatomical convergence centre has as yet been demonstrated. According to Heddeaus, destruction of the left sphincter nucleus with the fibres coming to it and going from it causes paralysis of the pupillary light reflex in that eye, but does not cause paralysis of the convergence reflex. Division of the third nerve abolishes the convergence reflex. This makes it probable that the impulses necessary for the production of the pupillary light reflex and convergence reflex are conducted along separate fasciculi of nerve-fibres coming from separate centres.

Heddaeus holds that this reaction is synergistic with convergence. The remarkable experiments of Marian [18] show conclusively that the reflex is not necessarily associated with contraction of the interni, but do not disprove the necessity for the integrity of the convergence action of muscles innervated by the third for its manifestation. Marian, operating on the eyes of monkeys, transplanted the tendon of the superior oblique to the insertion of the internus and the tendon of the internus to the insertion of the superior oblique. Divergence was the immediate result, but subsequently the eye could be made to converge with its fellow, showing that the convergence does not depend on the integrity of the internus. Again, the place of attachment of the internus was exchanged with that of the externus of the right eye. On movement of both eyes to the right (contraction of both interni) there was no contraction of the pupils. The externi were made to take the place of the interni and eventually convergence became possible. This was accompanied by contraction of the pupil, showing that the centre for the interni in the nucleus of the third has no intimate connection with the centre for the convergence pupillary reflex. The experiments do not disprove the connection of the superior and inferior recti, which undoubtedly assist in convergence, with the convergence pupillary reflex. Consequently, Marian's conclusion to that effect, although possibly correct, is not warranted. These experiments serve to emphasize the fact that little is known of the production of the convergence pupillary reflex. Marian thinks it possible that an inhibiting impulse may affect the dilator centre and permit of a passive contraction of the sphincter. The opinion held by some observers that the convergence pupillary reflex depends on an impulse originating in a *cortical* centre simultaneously with the voluntary impulse for convergence operating through the intermediate nucleus of the third nerve may be correct. Loss of convergence pupillary reflex in one or both eyes has been observed without loss of convergence.[19] This fact implies a centre acting either directly to cause contraction of the sphincter pupillæ or indirectly to inhibit (Marian) the action of the sympathetic on the dilatator pupillæ.

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[18] Deutsch. Zeitschr. f. Nervenheilk., 1903, Bd. xxiv., S. 274.

In affections of convergence light reflex, the following condition may obtain:

(a) Paralysis of convergence light reflex on one side without other paralyses; retention of pupillary light reflex direct and consensual, on both sides. The pupil on the affected side is usually larger than normal by strong illumination.

(b) Paralysis of convergence light reflex on both sides without other paralyses (other reflexes remaining intact).

Lesions causing paralysis of the third in any part of its course cause loss of the convergence pupillary reflex.

Sympathetic Dilator Tract.—The fibres which innervate the dilatator pupillæ have their origin in the ciliospinal centre of the spinal cord, situated in the anterior cornu[20] in the lower part of the cervical and upper part of the dorsal cord. According to Bach, this centre is connected with the sphincter nucleus of the third. A centre in the medulla is supposed by Bach to be inhibitory to both of the centres with which it is connected, namely, with the ciliospinal centre and the sphincter nucleus of the third. Ruge[21] repeated the experiments of Bach on rabbits and found that after complete division of the medulla oblongata perceptible pupillary reaction could be obtained, apparently conclusively disproving Bach's idea of an inhibitory pupillary reflex centre in the medulla.

Fibres from the dilator centre in the cord pass by the rami communicantes which pass out of the spinal canal with the seventh and eighth cervical and first and second dorsal nerves, and enter the cervical sympathetic. The motor fibres reach the superior cervical ganglion, from which similar fibres pass to the internal carotid plexus; thence to the Gasserian ganglion and in the superior division of the fifth into the nasal branch and by way of the long ciliary nerves to the globe. These nerve filaments pierce the sclera near the optic nerve and pass between sclera and chorioid forward into the ciliary body, the outer layers of which they pierce, reaching the iris.

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[19] The writer has recently observed a case in which, with double choked disk accompanied by headache, there was preservation of the pupillary light reflex, both direct and consensual, no paralysis of accommodation or of the power of convergence, but complete loss of convergence pupillary reflex. At the same time there was paresis of the external rectus of the right eye. Under mixed treatment the eyes resumed the normal condition. This would point to a convergence centre entirely separate from the sphincter or accommodation nucleus.

[20] Coppez, Revue génér., February 28, 1903.

[21] Graefe's Archiv, Bd. liv., H. iii., S. 483.

It is probable that an interruption of the pupillo-dilator fibres takes place in the superior cervical ganglion, as stimulation by non-electrical agents produces dilatation of the pupil, which does not occur on irritation of non-sensitive tissues unless the faradic current is employed.

Anderson,[22] experimenting on cats, after excision of the *ciliary ganglion*, also after section of the short ciliary nerves which he considers equivalent to section of the third nerve (motor oculi), could not obtain a *contraction* of the pupil by light or by any form of stimulus, but obtained marked *dilatation* of the pupil (1) by stimulating the proximal end of the sciatic nerve by means of the faradic current; (2) by pinching the skin; (3) by stroking the hair.

The cord was divided at the level of the first cervical vertebra and the medulla and brain destroyed. Faradic stimulation of the proximal end of the sciatic nerve produced distinct dilatation after a one per cent. solution of eserin had been instilled, demonstrating the fact that the dilatation could not be due to inhibition of the pupillo-constrictor tone. Section of the cord below the second dorsal nerves abolishes all dilatation reaction to stimuli applied below this point. These experiments, which have also been corroborated by others, prove that the dilator nerve impulses pass through the centre in the cervico-dorsal cord and through the upper cervical ganglion. Parsons[23] has shown that stimulation of the cortex of the brain has no effect on the pupil after division of the third and sympathetic nerves, but, if the sympathetic alone is divided, dilatation may be obtained, which Parsons explains as being due to an inhibitory effect on the third nerve.

The experiments of Budge,[24] Hensen and Volkers (1868), O. Langendorf (1864), and others prove that stimulation of the ciliary ganglion and short ciliary nerves causes contraction of the pupil, and that stimulation of the long ciliary nerves causes dilatation of the pupil. Piltz's experiments demonstrate the fact that the contraction of the pupil resulting from stimulation of the short ciliary nerves comes on suddenly and subsides suddenly on removal of the stimulus; that on stimulation of the long ciliary nerves the dilatation comes on in about two seconds and subsides very slowly on removal of the stimulus.

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[22] Journal of Physiol., 1903. 30.

[23] Ibid., 1901, xxvi. p. 366.

[24] Die beweg. d. Iris, Braunschweig, 1855.

The effect on the pupil by stimulation of the sympathetic is to produce marked dilatation of the pupil on the *side affected,* which is greater than the dilatation after abolition of the function of the third nerve, but is not so great as is obtained by the use of potent mydriatics. The dilatation after division of the third on stimulation of the sympathetic reaches almost, if not quite, maximum dilatation, showing the effect of the contraction tone of the sphincter under normal conditions of the third. Section of the cervical sympathetic, removal of the superior cervical ganglion or section of the long ciliary nerves produces contraction of the pupil on the side affected without entirely abolishing the pupillary light reflex. The miosis is maximal.

Braunstein's experiments[25] caused him to conclude as follows:

1. Stimulation of the outer of the two long ciliary nerves causes a dilatation of the pupil in the outer upper half.
2. Stimulation of the ocular end of the divided cervical sympathetic nerve produces an even dilatation of the pupil, not maximal.
3. Stimulation of the divided cervical sympathetic after division of the outer of the two long ciliary nerves produces dilatation of the inner lower half of the pupil (Compare Chapter I, pp. 5, 8).

EXAMINATION OF THE LIGHT REFLEX.

If the eye, after being covered, is quickly uncovered and exposed to moderately bright illumination, the pupil rapidly contracts and reaches its minimum size (primary narrowing). There is then a slight dilatation (secondary dilatation) and perhaps a second slight contraction before the size becomes fixed. This phenomenon may be even more plainly manifest in the change from parallelism of the visual axes (distance vision) to strong convergence (near vision). The oscillation of the iris under these normal conditions resembles in small degree the phenomenon known as "hippus." The primary contraction may occur very quickly; indeed, so quickly that it may not be noticed, the second dilatation being the first movement seen. Under these circumstances the movement may be readily mistaken for the "paradoxical" pupillary reaction.

Examination for the Direct Light Reflex.—If there is any doubt regarding the reaction of the pupil to light, the following method should be employed. The patient should be placed in a dark room facing and "fixing" a single source of light. The light should be not more than one metre from the patient. One eye of the patient is excluded from the light by covering it with the hand or some opaque object. By means of a convex lens (15 to 20 D.) the light is now focussed in the exposed eye, the patient not being permitted to look in any other direction than in a direct line to the light. The light may be thrown into the eye and shut out of the eye at the will of the examiner. The position and direction of regard eliminate any influence upon the pupil by accommodation or by convergence. The strong illumination of the eye thus obtained makes it possible to determine with certainty the presence or absence of the pupillary light reflex. Points to determine are:

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[25] Ein Beiträge z. Kent. d. Innervation d. Irisbewegungen, Charkow, 1893.

(1) **Are the pupils of normal size when the eyes are in shadow and in light?**

(See normal pupil.) The pupils may be larger than normal and of equal size *in shadow* if under the influence of a mydriatic. They may be smaller than normal and equal in size in light (a) if the irides are equally inflamed; (b) if they are atrophic; (c) if there is disease of the sympathetic affecting both sides; (d) in senile atrophy; (e) if under the influence of a miotic.

The pupils may be larger than normal *when exposed to bright light* (a) in loss of central acuity of vision and in total blindness due to a lesion peripheral to the corpora quadrigemina; (b) when there is a lesion involving the centripetal fibres of the pupillary light reflex arc (page 292); (c) in cases of glaucoma with increased tension; (d) when belladonna or other mydriatics are being taken internally in sufficient quantity or when mydriatics have been instilled into the eyes; (e) when paralysis of the pupillary constrictor fibres exists, or (f) when there is a lesion of the pupillary light reflex arc at any point.

The pupils may be smaller than normal *when exposed to bright light* (a) when miosis has been induced by a miotic remedy, as by eserin, pilocarpin, etc.; (b) when the sympathetic on one or both sides is paralyzed; (c) in some cases of inflammation affecting both irides.

(2) **Are the pupils of equal size in dilation and in contraction?**

The pupils will be of unequal size *in shadow*, the pupil of the affected eye being larger (a) when a mydriatic has been used in one eye only; (b) when there is paralysis of the centripetal fibres of the pupillary light reflex arc (Fig. 6); (c) in disease affecting the tissues of the irides unequally. In paralysis of the dilator tract (see Fig. 6) affecting one side the pupil of the non-affected eye will be the larger.

The pupils will be of unequal size in contraction *by bright light*, being larger (a) when a mydriatic has been used in one eye; (b) when the centripetal light reflex tract on one side has been interrupted; (c) in glaucoma with plus tension, affecting one side more than the other; (d) temporarily, following a blow on the eye which has produced paresis of the sphincter pupillæ.

The pupil will be smaller than normal (a) when a miotic has been used in one eye; (b) in paralysis of the centripetal dilator tract affecting one eye more than the other; (c) in some cases of congestion of the iris of one eye.

Examination for the Indirect or Consensual Light Reflex.—The patient may be placed facing a window, one eye being screened in such a manner that it will be in shadow, but so that the observer may see the iris sufficiently distinctly to note its movements. The exposed eye is then alternately covered and uncovered, the movements of the iris of the screened eye being observed. On excluding the light from the unscreened eye, the pupil of the screened eye should dilate. On re-admitting the light to the unscreened eye the pupil of the screened eye should contract. The indirect or consensual reaction should equal the direct reaction.

Instead of using daylight, the patient may be placed in a moderately-lighted room with both eyes exposed and the direct reaction be excited by reflecting light into one eye by means of a mirror from a source of light placed behind the plane of the patient's face.

The consensual light reflex may be diminished in both eyes (a) when the visual acuteness of both eyes is much diminished by disease affecting retina, optic nerve, chiasm, or tractus; (b) when the centrifugal dilator tracts are partly, but not completely, paralyzed; (c) when there is disease of the parenchyma of the irides; (d) when mild mydriatic or miotic remedies are instilled into the eye or taken internally in sufficient quantity; (e) when the tension of both eyes is slightly elevated.

The consensual light reflex will be *lost* in both eyes (a) when there is complete blindness due to disease affecting any part of the optic tract distal to the point where the centripetal fibres of the light reflex arc leave the tract (see Light Reflex Arc); [26] (b) when there is paralysis of the centrifugal dilator tract on both sides at any point; (c) when the sphincter pupillæ or dilatator pupillæ muscles are paralyzed by the use of mydriatics or miotics; (d) when the tension is above normal in both eyes.

The consensual light reflex may be diminished in one eye under the same conditions as in diminished light reflex of both eyes when one eye or the tracts on one side are affected.

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[26] In complete blindness due to double cortical homonymous hemianopsia the pupillary light reflex is preserved.

The consensual light reflex will be diminished in the *normal* eye (a) when impairment of vision is due to a lesion of the opposite tract distal to the point where the centripetal light reflex fasciculus leaves the optic tract. In the *affected* eye (a) when there is a mild effect from the instillation of mydriatics or miotics; (b) when partial paralysis of the dilator tract exists at any part of its course; (c) when paresis of the sphincter nucleus or sphincter fasciculus of the third nerve exists; (d) when the tension of the eye is greatly increased.

The consensual light reflex may be *lost in one eye* (a) when complete paralysis of the sphincter pupillæ from mydriatics or a blow occurs; (b) when the sphincter nucleus or sphincter fasciculus of the third nerve is paralyzed and when great increase of tension affects one eye.

In some cases **examination by means of photography** is desirable, particularly, as Schaefer [27] has pointed out, in the cases of anisocoria without iridoplegia, when almost maximum dilation of both irides simultaneously is recorded.

The Light Reflex, Direct and Indirect (Consensual), Excited by Illuminating the Various Parts of the Retina.—To test the light reflex of *various parts* of the retina, it is necessary to arrange the illumination so that with the patient in diffuse light of sufficient brightness to permit the observer to discern the movements of the pupils, a pencil of light of sufficient intensity may be thrown into the pupil, so directed that only the part of the retina to be tested will be illuminated. It is difficult to illuminate a particular part of the retina without more or less diffusely illuminating other parts, thus making the results of the test unreliable. The rays of light should be so directed that they will come to an approximate focus on the retina, the refractive error of the patient being taken into consideration. The size of the incident cylinder of rays should be a little smaller than the pupil. The instrument of von Fragstein and Kempner (Fig. 7) may be employed and if properly made is very satisfactory.

Heddaeus advises the use of a very weak source of light, as weak as can be employed and enable the observer to see the reaction (a candle- or lamp-flame), reflecting the light into the eye by means of the plane mirror. In hemianopsia the light should be thrown on the retina as peripherally as possible, having the patient rotate the eyes to favor this, in order that the sensitive part of the retina may not be illuminated and the reflex excited. If the rays of light as they enter the eye are parallel or nearly so, the part of the retina most intensely illuminated will be very small and the best results will be obtained. The Haab reflex (page 304) may be employed as a control measure.

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[27] Dissertation, Giessen, 1899.

The direct pupillary reflex will fail when light is thrown on that part of the retina corresponding to an absolute scotoma in the field of vision, occasioned by disease of the retina or disease of the visual tract *distal* to the cerebral end of the pupillary light reflex arc; that is, in cases of disease of the optic nerve, chiasm, or tractus. The pupillary light reflex may be diminished in cases in which the blindness is not quite complete (the scotoma not being absolute).

In cases in which the scotoma is absolute, but the lesion in the visual tract is situated *proximal* to the cerebral end of the pupillary light reflex arc, other conditions being normal, the illumination of the corresponding blind part of the retina will elicit the direct and consensual pupillary light reflex (Wernicke pupillary reaction).

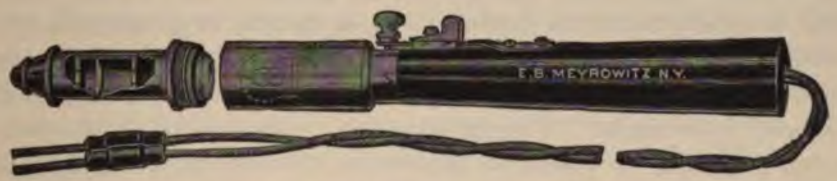

FIG. 7.—Von Fragstein and Kempner's apparatus for testing the pupillary reaction.

Method of Testing the Convergence Reaction.—The individual is first directed to look at some object at a distance, when the size of the pupil is noted. He is then requested to look at a point at a distance of ten or twelve inches from the eyes and in the median plane of the head, the illumination remaining unchanged. When the gaze is changed from the distant to the near-point a contraction of the pupils will occur. If now the gaze is again directed to the distant object, a dilatation of the pupils will take place. This reaction will take place whatever the degree of illumination. If a card of test-type is held before the eyes and removed and approached to the eyes, an excellent convergence pupillary reflex may be obtained. Individuals who cannot converge and accommodate (small children, the blind, and the insane) do not show the convergence pupillary reflex.

Sensory Reflex (Pain Reflex).—Dilatation of the pupil is excited under normal conditions by irritating sensory parts in almost any part of the body. The reflex path is by way of the sensory nerves to the ciliospinal centre in the cord and along the dilator tract to the iris. If the skin of the neck, particularly the back of the neck, is pinched, pricked, or pulled, the palm of the hand or sole of the foot tickled, dilatation of the pupil follows.

The diameter of the pupil changes (dilates) as a result of every emotion—fright, fear, sudden joy or sorrow, amusement. Bumke[28] attributes this to excitation of the cells of the cortex cerebri.

Irritation of the skin by sudden applications of cold or heat will cause the pupil to dilate.

It has been observed[29] that irritation or stimulation of the pneumogastric, hypoglossus and laryngeal nerves causes dilatation of the pupil.

Oculo-pupillary reflex is a term given by von Varady and others to the pupillary reflex due to exciting sensory surfaces, particularly of the skin and mucous membrane of the lids. Sensory mydriasis is probably always followed by comparative miosis. If the excitation is continued, mydriasis is again induced, followed by miosis. Von Varady found that the most practical method was to prick the skin of the eyelid with a needle and then observe the pupillary reflex, which is that of dilatation followed by miosis. As studied by von Varady, the reflex differed in different individuals. It might be absent in apparently healthy individuals, present or absent in functional disturbances or sensibility, namely, hysterical anæsthesia or hyperæsthesia, but it was never present in cases of organæsthesia, affording a diagnostic sign in this condition.

Haab's Cortical Pupillary Reflex (Attention Reflex).—In 1885 Haab (Sitzungbericht d. Ges. d. Aerzte in Zurich, Nov. 21) described a reaction of the pupil observed under the following conditions: If, when the individual is in a dark room, his line of vision being directed forward into space or against a dark wall, a lamp or candle is placed at a few feet or yards distance (1 metre) at an angle of 45° to the patient's line of vision and on the horizontal plane, the reflex will be excited, provided the *attention* (not the line of vision) can be directed to and held upon the flame. The reaction is a marked contraction of the pupil which is maintained as long as the attention remains fixed on the flame, but is relaxed whenever the attention is relaxed. The reflex is marked in individuals whose irides are freely responsive to the stimulus of light. Some practice is often necessary to elicit the reflex. When the power to fix the attention as described is acquired, other luminous sources beside the lamp (small window, slit in a blind, etc.) may be substituted if desired. Auto-examination may be conducted.[30] The degree of contraction of the pupil is in direct proportion to the degree of the illumination. If a piece of white paper, illuminated by a source of light not visible to the patient, be substituted for the lamp, the contraction of the pupil is less. If a piece of black paper or other black object be substituted, there is no contraction.

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[28] Centralbl. f. Nerv. u. Psych., October, 1903, p. 40

[29] Kowalewsky, Investigations on the Innervation in Dilatation of the Pupil, Kassan, 1885.

[30] Haab, Archiv f. Augenheilk., Bd. xlvi.

Variation in the position of the source of illumination causes variation in the degree of the contraction of the pupil. If the flame is carried backward beyond 45° on the horizontal plane, letting the point of fixation on the line of vision be represented by zero, the contraction becomes less; if in advance of 45°, greater. The reaction also becomes less marked if the source of illumination is above or below the horizontal plane.

Both pupils are affected equally with the light falling in one eye.

The reflex apparently depends on the normal condition of the visual fibres throughout corresponding to the side of the retina on which the light falls and on the normal condition of the centrifugal fibres from the cortex that are concerned in this reflex. According to Haab, the centripetal pupillary light reflex fibres, central from the chiasm at least, are not necessary (may be destroyed) for the production of this reflex.

Cortex Reflex.—Stimulation of the cortex has been found to produce an influence on the pupil, causing moderate dilatation. Many investigators have noted the fact that stimulation of the cortex of the brain, particularly of the occipital lobe, would cause dilatation of the pupil. Stimulation of the anterior four-fifths of the cortex of the cerebellum has caused dilatation.

Parsons,[31] experimenting on cats, dogs, and monkeys, found that electrical stimulation of the cortex of the brain " in the neighborhood of the crucial sulcus and over a considerable area of the occipital region" produced dilatation of the pupil. When the cervical sympathetic was intact the pupils became widely dilated, producing " all the usual effects of excitation of the nerves themselves." When the sympathetic was destroyed the dilatation was not abolished, but was only half or two-thirds as great as when the sympathetic was intact. This fact was strongly brought out when only one sympathetic was destroyed, the difference in the degree of dilatation of both pupils being very pronounced. With division of the sympathetic and of the third nerve, stimulation of the cortex fails to affect the pupils; they are immobile and remain one-half to two-thirds dilated. These experiments lead Parsons to the conclusion that cortical stimulation produces dilatation of the pupil both by stimulation of the sympathetic and by inhibition of the sphincter centre when the cervical sympathetic is intact, and by inhibition of the sphincter centre when the cervical sympathetic is destroyed.

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[31] Royal London Hospital Reports, June, 1904, p. 20.

Braunstein [32] obtained similar results, but attributed them wholly to inhibition of the third nerve.

Evidence of obtaining contraction of the pupil by stimulating certain parts of the cortex is not wanting. Schaefer induced strong contraction of the pupil by stimulating the quadrate lobule in monkeys, and Terrier by stimulating the middle of the convexity of the hemispheres of the brains of pigeons.

Changes in the size of the pupil by the will is claimed by some to be possible. I have myself observed a patient who, while standing before a window, looking forward into space, caused his pupils to dilate and contract. However, it is so difficult to dissociate accommodative effort in these cases that it is not possible to state definitely that these movements of the pupil are purely voluntary.

Orbicularis or Lid Reaction (Gifford-Galassi; Westphal-Piltz).—This was first described by Von Graefe [33] as an accompaniment of a general contraction of ocular muscles excited by contraction of the orbicularis. The reaction has since been described by many writers. By forcibly closing the eye it often occurs that the pupil contracts. If the lids are held open by a speculum or by other means and an attempt is made to close the lids, the pupil in many individuals will contract.

Harold Gifford, in writing of this reflex, expresses the opinion that it is the result of an overflow stimulus passing from the nucleus of the facial to the nucleus of the third nerve, along the longitudinal fasciculus. This view is also held by other writers. The reflex may be obtained in normal individuals, but often fails. As a rule, in cases of tabes with failure of reaction to light but with retention of convergence and accommodation reflex, the orbicularis reflex is present and is often more energetic than the convergence reflex (Westphal and others). Uhthoff has observed cases in which, with preserved convergence reflex, the orbicularis reflex has failed, and, conversely, cases with loss of convergence reflex in which the orbicularis reflex was preserved. Piltz found the orbicularis reflex preserved in 41 to 43 per cent. of the cases of tabes. Similar observations have been made in cases of progressive paralysis.

Gifford [34] writes that the reflex is most marked in individuals whose vision is poor and in eyes that are almost blind. While he does not ascribe much of diagnostic value to the reflex, he suggests that it may be of use to determine whether the sphincter is paralyzed as in cases of disease of the nucleus of the third nerve or in brain tumor; also possibly to determine whether a dilatation of the pupil is due to a mydriatic in doubtful cases. Gifford is of the opinion that the reaction depends chiefly on the palpebral portion of the orbicularis palpebrarum muscle and that if, as Mendel believes, the nerve supply to this part of the orbicularis comes from the posterior part of the third nucleus, the influence on the sphincter pupillæ is easily explained.

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[32] Zurm Lehre v. d. Inner. d. Pupillenbewegung, Braunschweig, 1894.

[33] Graefe's Archiv, Bd. i., H. 1, p. 318.

[34] Archives of Ophthal., vol. xxiv. p. 402.

Pupillary Reflex on Movement of the Eyes.—The size of the pupil is reduced in many cases by forcible abduction. Uhthoff [35] has observed this reaction in some cases of Argyll-Robertson pupil when the patient rotated the eye outward forcibly. Contraction of the pupil by forcible rotation outward has been observed by other writers. No pathological or diagnostic importance has been attributed to it.

PATHOLOGICAL VARIATIONS IN THE PUPIL.

The possible pathological variations in the pupil may be stated as follows (Uhthoff):

1. Loss of the pupillary light reflex with retention of the convergence and accommodation reflex. (Argyll-Robertson pupil.)
2. Loss of convergence and accommodation reflex and retention of light reflex.
3. Loss of the pupillary reflex for light, convergence, and accommodation.
4. Ophthalmoplegia interna, loss of all reflex movements of the pupil.
5. Loss of direct light reflex in an amaurotic eye and consensual light reflex in the fellow-eye, due to loss of vision from a lesion peripheral to the chiasm ("reflextaub," Heddaeus).
6. Loss of sensory or psychical reflex action.
7. Abnormal miosis with retention of light and convergence reflexes, either from abnormal stimulation of the sphincter pupillæ, which may include the so-called *myotonic pupillary* reaction, or from paralysis of the dilatator pupillæ.
8. Abnormal dilatation of the pupil with retention of the light and convergence reflex, stimulation of the dilatator pupillæ (spastic mydriasis).

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[35] Graefe Saemisch, 2 Aufl., Bd. xi., H. ii., p. 242.

9. Anisocoria (difference in size of pupils).
10. Changing anisocoria.
11. Irregular form of the pupils.
12. Hippus.
13. Paradoxical pupillary reflex.
14. Hemianopic pupillary reaction.

Argyll-Robertson Pupil.—Loss of the pupillary light reflex, either with miosis (spinal miosis, paralysis of the sympathetic, absence of dilator nerve impulse) or without miosis (sympathetic not paralyzed, skin reflex retained), constitutes Argyll-Robertson pupil. Both irides are similarly affected in the greater number of cases, but in some cases (13 per cent., Rochon-Divignand and J. Hettz) it is unilateral for a long time. In quite a large percentage of cases the pupillary light reflex is lost in one eye and only partly lost in the other.

Miosis is present in almost all tabetic cases.

Paralysis of accommodation of one or both eyes may accompany the Argyll-Robertson pupil.

The convergence pupillary reflex is absent in a small percentage of the cases of loss of pupillary light reflex. Moeli puts it at 1 per cent.; Uhthoff states that the proportion between the cases with loss of pupillary light reflex only and those with loss of convergence light reflex also, is 3.5 to 1. Argyll-Robertson pupil has been observed most frequently in tabes, where, according to Gowers, it is present in 80 per cent.; Stern, 90 per cent.; Uhthoff, 67 per cent. of the cases. The pupils are of the same size in both eyes as a rule. Of the cases observed by Uhthoff the pupils were unlike in size in 25 per cent.

The Argyll-Robertson pupil never occurs in healthy individuals. In 166 cases of loss of pupillary light reflex, Uhthoff[36] found 67.6 per cent. associated with tabes, 8.8 per cent. with dementia paralytica, 8.17 per cent. with syphilis without tabes, 5.87 per cent. with other cerebral diseases, 1.4 per cent. with multiple sclerosis, 2.0 per cent. with railway spine and injury of the head, 0.7 per cent. with congenital feeblemindedness, 0.7 per cent. with hystero-epilepsy, 0.7 per cent. with abuse of tobacco, 0.7 per cent. with hemianæsthesia dextram, 3.6 per cent. with no assignable cause. It has been observed in hypertrophic peripheral neuritis (Dejerine), in polyencephalitis hæmorrhagica, and in polyneuritis alcoholica.

Uhthoff[37] has collected the statistics of Moeli, Siemerling, and

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[36] Berlin. klin. Woch., 1886, No. 3.

[37] Graefe Saemisch, Handbuch, 2d ed., chap. xxii. p. 223.

Thomsen, embracing 4000 cases of diseases of the nervous system (inmates of asylums). Absence of pupillary light reflex was found in 492, distributed as follows: Progressive paralysis, 85.5 per cent.; tabes, 4.25 per cent.; syphilis, 1.83 per cent.; multiple sclerosis, 1.2 per cent.; injury to head, 0.6 per cent.; paranoia and epilepsy, 2 per cent.

Loss of pupillary light reflex is, as a rule, permanent, since it occurs the greater number of times as an accompaniment of diseases of the nervous system that are progressive. It is sometimes present in cases of disease of the nervous system which tend to recover; under these circumstances the pupillary light reflex may be re-established. Sulzer[38] observed loss of pupillary light reflex in the early stage of syphilis in 11 of 53 cases examined. He followed the pupillary anomaly in some of the cases, ascertaining that the condition may develop within three months after the primary sore. He states that in general the loss of pupillary light reflex in such cases is transitory.

It is held by some writers that the Argyll-Robertson pupil is intermittent in some cases. Tanzi[39] reports such a condition in a case of progressive paralysis. Uhthoff is of the opinion that it does not occur in tabes, but thinks that it may occur in less confirmed pathological conditions of the nervous system.

Loss of pupillary light reflex is recognized as often being a very early symptom of tabes.

Dejerine,[40] Dufour,[41] and Uhthoff are of the opinion that all cases of Argyll-Robertson are due to syphilis. This view is combated by others. Jaffray has observed it in aortic disease. P. Maria regards this form of pupil as an evidence of disease of the posterior columns of the cord which may or may not be syphilitic.

Seat of the Lesion.—It has been stated that one-sided loss of pupillary light reflex occurs and that in certain cases loss of convergence reflex and loss of the power of accommodation occur; also that miosis is most frequently present in loss of pupillary light reflex. Heddaeus is of the opinion that the lesion in cases of one-sided loss of pupillary light reflex affects the centrifugal fasciculus of nerve-fibres which proceed from the sphincter nucleus, the fasciculus for the accommodation reflex remaining uninjured. If the fasciculus from the accommodation nucleus is involved, there is total one-sided iridoplegia. The consensual reaction of the fellow-eye is not affected. Others regard the one-sided loss of pupillary light reflex as a nuclear affection. Uhthoff is of the opinion that lesion of the portion of the centripetal pupillary light reflex fibres lying between the tractus or external geniculate body and the sphincter nucleus has more in its favor. If there is a lesion of this bundle on both sides at this point, the light reflex will be abolished. Miosis might be produced by irritation of the sphincter nucleus, an extension of the process to this centre, but Uhthoff believes that the miosis is due to a lesion of the cervical sympathetic.

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[38] Ann. de Dermatol. et de Syph., March, 1901.

[39] Riv. di patol. nerv. e. ment., iv. S. 385.

[40] Soc. de Neurologie de Paris, June 15, 1902.

[41] Bull. de la Soc. méd. des Hôpitaux de Paris, January 13, 1902.

Pathological research in cases of loss of pupillary reaction has afforded but little light on the subject. Pincles, Zeri, and others, in examining microscopically the brains of tabetics, found pathological changes in the floor of the aqueduct of Sylvius. Round-cell infiltration was found in this locality by Kostenitsch. In the examination of 20 cases of progressive paralysis with Argyll-Robertson pupil, Schutz[42] found pathological changes in the nerve-fibres in the gray substance in the floor of the aqueduct (sclerosis) to which he attributed the loss of the pupillary light reflex; however, he was not able to demonstrate any particular bundle of nerve-fibres as being concerned in this degeneration. Siemerling and Boedeker have met with similar changes, also with minute hemorrhages in this tissue. The cases of Moeli (page 291) and Monakow, who found in a case of one-sided loss of pupillary light reflex a small sclerosed focus in the tissue between the sphincter nucleus and the external geniculate body, apparently involving the centripetal pupillary light reflex bundle as it passes from the tractus to the sphincter nucleus, support Uhthoff's position.

A number of investigators refer the lesion to the cervical portion of the spinal cord. Wolf[43] found that in cases of tabes and also general paralysis in which the light reflex was lost and the knee-jerk intact, the lesion affected the posterior columns of the cervical cord. In cases in which the pupillary light reflex was retained and the knee-jerk was lost, the cord was affected below the third or fourth dorsal vertebra. In cases in which both pupillary light reflex and knee-jerk were lost, the entire cord was affected. Others have made similar observations. These investigators have failed to examine the brain tissue in the floor of the third ventricle to determine whether it was or was not involved. Cases of loss of pupillary light reflex with intact cord have been reported (Dejerine) and cases of cervical tabes with retained pupillary light reflex have also been reported (Buck).

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[42] Archiv f. Psych., Bd. xxii., H. 3, S. 527.

[43] Ibid., xxxii., H. i., S. 57.

Recently Marina[44] has examined the ciliary ganglion and ciliary nerves in cases of loss of pupillary light reflex and claims to have found pathological changes in all of the cases. He is of the opinion that the seat of the lesion is in these structures.

Ruge advances the hypothesis that disease of the ciliospinal centre is followed by atrophy of sympathetic nervous elements in the ciliary ganglion, causing an interruption in the reflex arc. The reflex iridoplegia of tabes is brought about in this way, making a double lesion unnecessary to explain the phenomenon.

Clinical and pathological evidence regarding the site of the lesion in loss of pupillary light reflex to the present time fails of positive localization. The weight of evidence indicates a lesion in the vicinity of the nucleus of the third and the occasional accompaniment of paralysis of extrinsic eye-muscles as well as the ciliary muscles makes the supposition plausible.

Loss of Convergence Accommodation Reflex and Retention of Light Reflex.—This condition is rarely met with. It occurs in most cases of congenital absence of convergence, in some cases in which the power of convergence is lost; also in some cases in which the convergence is abandoned, as in loss of the vision of one eye or in conditions in which one eye is not used. It has been observed in tabes, but must be extremely rare, as Uhthoff writes that he has never seen it. The author has observed it in syphilitic basal meningitis, and it has been seen in progressive paralysis, myelitis, and tumor of the corpora quadrigemina.

The site of the lesion is supposed to be in the brain tissue immediately below the sphincter and accommodation nucleus, so placed as to involve the fasciculus of fibres which, according to Heddaeus, joins the convergence centre with the oculomotor nerve.

Loss of convergence pupillary reaction may affect one pupil alone or both pupils. The latter is by far the more common. The convergence reaction is seldom absent in tabes, but it occurs in some cases. Sulzer[45] reports two cases of absence of convergence and light reflex in 53 cases of early syphilis. Moeli[46] reports it in one per cent. of 500 cases examined. Uhthoff[47] found absence of the convergence pupillary reflex in syphilis, 8 cases; tabes, 3 cases; progressive paralysis, 2 cases; injury, 2 cases; "cold," 1 case; tuberculosis of the brain, 1 case; tumor of the brain, 1 case; no assignable cause, 12 cases. Convergence pupillary paralysis has been observed also after diphtheria, alcohol intoxication, tuberculosis, Basedow's disease.

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[44] Ann. d. Neurologie, vol. xix. p. 209.

[45] Ann. de Dermatol. et de Syph., March, 1901.

[46] Centralbl. f. prakt. Augenheilk., 1885, p. 272.

[47] Berlin. klin. Woch., 1886, No. 3.

In some of the cases in which convergence and accommodation pupillary reaction is lost, mydriasis is present, even when the power of accommodation is preserved.

Ophthalmoplegia interna is a term given to the condition of isolated paralysis of the sphincter pupillæ and the ciliary muscles without involvement of the extrinsic eye-muscles. The lesion in these cases is without doubt nuclear. It is most frequently unilateral, but may be bilateral. Not infrequently the sphincter will be involved first, the ciliary muscle later; the reverse may occur. In rare cases there may be isolated paralysis of one or the other muscle.

In regard to the cause, Uhthoff has collected 30 cases. Syphilis was the cause in 23.3 per cent.; tabes, 10 per cent.; progressive paralysis, 6.6 per cent.; traumatism, 6.6 per cent.; cold, 3.3 per cent.; intracranial disease, 6.6 per cent.; cause unknown, 40 per cent. The proportion of unilateral to bilateral was approximately 2 to 1. Syphilis is by far the most frequent cause. In six cases observed by the writer a history of syphilis was obtained in all, and in all the primary syphilitic lesion had occurred many years before. Post-diphtheritic ophthalmoplegia interna has been observed.

In quite a high percentage of cases other portions of the nucleus of the third nerve and the nucleus of the fourth and sixth nerves become involved. The condition of partial or complete ophthalmoplegia externa develops. At times paralysis of the intrinsic muscles follows paralysis of the extrinsic muscles.

Prognosis.—Unilateral ophthalmoplegia interna may remain for many years unchanged, but the greater number of cases recover partly or wholly or are complicated by involvement of other parts of the central nervous system. Double ophthalmoplegia interna is not as a rule so apt to remain unchanged.

Loss of sensory or psychical reflex action will occur in any case in which there is a lesion affecting the dilator pupillary tract. Destruction of the ciliospinal centre, with consequent miosis; of the superior ganglion of the cervical sympathetic, which produces the symptom complex of ptosis, miosis, enophthalmus, and reduction in the tension of the globe; of the centrifugal dilator tract distal to the ganglion with the consequent miosis, all produce loss of sensory and psychical reflex, without abolishing the light reflexes.

Loss of the sensory reflex occurs in all cases of tabes with miosis and Argyll-Robertson pupil. It is in fact absent in all individuals who from any cause present miosis with loss of pupillary light reflex. It may also be absent in cases of miosis with retention of the light reflex. When this occurs, lesion of the dilator tract in some part is the cause. Aside from tabes, this condition occurs in progressive paralysis, some cases of syphilis, multiple sclerosis, and traumatism.

This reflex is retained in all normal individuals and in those without miosis in whom the pupillary light reflex is retained.

In cases of miosis with loss of sensory reflex, mydriatics effect a partial dilatation of the pupil, probably by direct effect on the dilator muscle-fibres.

Abnormal miosis with retention of light and convergence reflex may occur from abnormal stimulation of the sphincter pupillæ or from paralysis of the dilatator pupillæ (paralysis of the sympathetic).

Stimulation miosis (spastic miosis) is frequently accompanied by spasm of accommodation. This may be brought about by hemorrhagic encephalitis, brain tumor, or abscess which does not involve the sphincter nucleus, but simply stimulates or irritates the neurons of this group of nerve cells. If the brain disease advances and destroys the sphincter nucleus, dilatation supersedes miosis. Spastic miosis may be due to the influence of light in individuals with hypersensitive retinæ, as in neurasthenia and in hyperemia of the retina and optic nerve, and, according to Heddaeus, in the first stages of alcoholic amblyopia, abnormal use of the eyes and as a result of excessive sexual indulgence. Excessive synergistic contraction of the pupils, occurring in hypermetropes who are obliged to converge excessively in order to accommodate sufficiently, especially in the early stage of presbyopia, may produce spastic miosis. This may also occur in emmetropic eyes in cases of weakness of the interni.

Except that produced by miotics, spastic miosis results in narrowing the pupil to a greater degree than anything else. The pupil contracts very slightly to light and accommodation and is influenced to some degree by miotics. It differs from paralytic miosis in that mydriatics cause dilatation *ad maximum*.

Cases of alternating spastic miosis and mydriasis have been reported by Bielschowsky,[48] in one of which alternating miosis and mydriasis occurred in the right eye at intervals of twenty to thirty seconds. There was right partial oculomotor paralysis. When mydriasis was present, there was slight divergence. When miosis came on, the eye turned inward and downward.

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[48] Klin. Monatsblatt f. Augenheilk., Bd. xvi. p. 308.

The myotonic pupillary reaction of Saenger[49] is closely allied to spastic miosis. This reaction is in the nature of a tonic spasm and may be excited by light, convergence and accommodation, and by the orbicularis reflexes. It is characterized by a very slow recovery from the miosis occasioned by the stimuli mentioned. When the stimulus is removed, the dilatation of the pupil, which normally follows the reflex miosis very rapidly, takes place slowly; that is, in from one-half to ten minutes.

The phenomenon has been observed only in patients with disease of the nervous system. Saenger observed it in a case of suspected tabes. The pupillary light reflex was lost. The patient was conscious of the excessive contraction of the pupil, he experienced a strained or cramped feeling in the temple and forehead. Distance objects were blurred at this time, indicating spasm of accommodation. Piltz has observed the myotonic pupillary reaction in general paresis with loss of pupillary light reflex.

Saenger is of the opinion that the site of the lesion is in the iris and that the condition is analogous to Thomsen's disease.

Abnormal dilatation of the pupil with retention of the light and convergence reflexes (spastic mydriasis) is met with in cases of stimulation of the cervical sympathetic, whether due to inflammatory processes affecting the ciliospinal centre or superior cervical ganglion or by sensory or psychical influences. It may accompany intracranial effusions and intracranial tumor.[50] Abnormal dilatation has been observed after epileptic seizures, premonitory to locomotor ataxia, in acute mania, and in melancholia. Bumke[51] observed excessive dilatation of the pupil in fifteen cases of catalepsy, with retention of light and convergence reflexes, in marked anæmia, and sometimes in imbeciles. It is often present in cases of intestinal irritation, as from helminths. The pupils are dilated and fixed in amaurosis, in complete paralysis of both third nerves, and when mydriatics have been used.

Anisocoria.—Difference in the size of the pupils is frequently observed. Slight differences may exist under perfectly normal conditions (page 288). Pronounced differences in the size of the pupils is pathological almost without exception. Difference in the size of the pupils, the abnormal pupil being *smaller* than its fellow, will occur in paralysis of the sympathetic affecting one side, when a miotic has been used, when there is congestion of the iris.

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[49] Neurolog. Centralbl., vol. xxi. p. 837.

[50] When in the course of intracranial disease of any kind, miosis gives way to mydriasis, the symptom is one of grave import, as it signifies partial or complete paralysis of the sphincter nucleus either by direct involvement or by excessive pressure; paralyses of other branches of the third nerve often accompany or follow this condition.

[51] Centralbl. f. Neur. u. Psych., October, 1903, p. 41.

The abnormal pupil will be *larger* than its fellow when there is stimulation of the sympathetic on one side, when a mydriatic has been used in one eye only, when there is paralysis of the centripetal fibres of the pupillary light reflex arc, after a blow that has produced paresis of the sphincter pupillæ, in increase of intra-ocular tension affecting one eye more than the other, in paralysis of the motor oculi, in disease of the nucleus of the third nerve affecting the sphincter portion.

Inequality of the pupils " may lead one to suspect locomotor ataxia, general paralysis of the insane, unilateral lesion of the third or of the sympathetic nerve, pain in a branch of the fifth nerve, carotid aneurism or tumor of the neck, unilateral cranial lesion, unilateral acute glaucoma."

A difference in the size of the pupils is common in tabes. Of 166 cases examined by Uhthoff, 25 per cent. presented a difference in the size of the pupils. In 25 per cent. of the cases of syringomyelia the pupils are uneven, due to involvement of the ciliospinal centre. Schauman[52] has noted a difference in the pupils of 723 subjects. The number includes 401 subjects, or 55.5 per cent., with no organic lesions but some neurosis, including 11 with exophthalmic goitre, 4 epileptics, and 3 with chorea. In the remaining 322, or 44.5 per cent., there were organic lesions, including tuberculosis in 6.8 per cent., and some vascular affection in 5.5 per cent. When anisocoria is not due to an organic lesion, it is the expression of an asthenic diathesis or a sign of hereditary degeneracy.

Irregularity of the Pupils.—Piltz[53] observed in a new-born child that the pupil was not circular, but that it soon became so. He concludes that synergetic action of the various parts of the iris does not obtain at birth, but soon asserts itself.

When irregularity of the pupil is observed, it is advisable to use a mydriatic for the purpose of excluding synechiæ. Irregularity of the pupil consists in irregularities in the margin of the pupil due to defective innervation of the various parts of the muscles of the iris. Thus lesion of the outer branch of the long ciliary nerves causes contraction of the upper outer half or quadrant of the iris and a flattening of the pupil in that part. The inner branch of these nerves supplies the lower inner half of the iris. The various nerve-fibres from the sphincter nucleus are distributed to the various parts of the sphincter pupillæ and lesion of a part of these fibres would cause paralysis of portions of the sphincter pupillæ only.

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[52] Abstract, Journal Am. Med. Asso., July 11, 1903, p. 143.

[53] Neurol. Centralbl., 1903, vol. xxii. p. 662.

Piltz divides irregularities of the pupil not due to synechiæ into two classes—(1) changing; (2) constant.

Changing irregularities present the following conditions. When the pupillary light reflex is tested, some parts of the iris contract while other parts do not. In a few days the part that was stationary is active, the other parts becoming stationary. Piltz has observed this form in progressive paralysis and in katatonia. He observed a case in which on the 21st of January, 1899, the right pupil was eccentrically displaced downward and outward; on the 24th of January the same pupil was displaced upward and outward. The pupillary light reflex was lost in both eyes and convergence reflex reduced. The orbicularis reflex was preserved to some degree. Piltz is of the opinion that changing paralysis is due to a shifting of the lesion in the nucleus.

Fixed irregularities are due to lesions in the nerves that innervate the iris that are more or less constant. Piltz observed seven cases of progressive paralysis, with irregular pupils in six. The inmates of a Swiss asylum for the insane showed irregular pupils in 42 per cent. Of 62 cases of tabes, 29 per cent. presented irregular pupils. Intraocular conditions not infrequently cause irregularity of the pupil, and intra-orbital conditions may cause injury of a long or short ciliary nerve or destruction of the ciliary ganglion, but these cases are very rare. Joffroy[54] states that within the last ten months preceding the writing of his article he had not seen a case of Argyll-Robertson pupil that did not show some irregularity of the margin of the pupil. He regards the condition as often the first sign of advancing disease of the innervation of the iris which eventually goes on to complete loss of the pupillary light reflex (Argyll-Robertson pupil) and in some cases to ophthalmoplegia interna. Piltz has observed that irregularities in the pupillary margin are of frequent occurrence in progressive paralysis; that in incipient progressive paralysis it is at times the first and only symptom; and it is sometimes the very first sign of the development of Argyll-Robertson pupil. The changes observed indicate that they are due to disease of the nerves supplying the iris or to their nuclei.

Hippus.—Under certain conditions, especially when both eyes are shaded from the light and then quickly uncovered, the patient being placed before a window or other source of light, the pupils will alternately dilate and contract. This change in size of the pupil under uniform illumination and without the influence of accommodation or convergence is known as hippus. It is a clonic spasm of the sphincter pupillæ (Fick). It may not be associated with impaired vision, but is occasionally observed in developing cataract. It is sometimes associated with nystagmus. It is also seen in neurasthenia, hysteria, epilepsy, disseminated sclerosis, cerebrospinal sclerosis, in acute meningitis, and in some cases of brain tumor. In observing the changes in the size of the pupil, it must be remembered that the normal oscillation differs much in different individuals. In patients with irides that react very readily to the stimulus of light, oscillation is more pronounced than in irides that react slowly. It is difficult to determine where normal oscillation leaves off and abnormal oscillation begins.

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[54] Rev. neurologique, 1902, p. 275.

According to Schrameck,[55] hippus occurs in cases in which there is paresis of the extrinsic ocular muscles, particularly when improvement is taking place. It has been observed in a case of absence of pupillary light reflex (Uhthoff), also in a case of double amaurosis (Ewitzky).

Paradoxical Reflex.—Piltz[56] classifies the paradoxical reflexes as follows: (1) Paradoxical reflex (*a*) on near vision, convergence dilation of the pupil; (*b*) contraction of the pupil in distance vision. (2) True paradoxical pupillary reflex, (*a*) dilatation by bright light; (*b*) contraction of the pupil when the light is withdrawn, with subdivisions (*aa*) rapid dilation without preceding contraction or (*bb*) pupil contracts without preceding dilation.

Experiments on rabbits and other animals have shown that section of the cervical sympathetic on one side causes constriction of the pupil, and removal of the superior cervical ganglion on the opposite side causes slightly greater constriction of the pupil on the same side, but under certain conditions, excitements, anæsthesia, dyspnœa, the pupil on the side of the excised superior cervical ganglion becomes more dilated than on the side of the divided nerve. This phenomenon has been termed paradoxical pupil-dilatation. It has been observed that the pupil on the side of the excised superior cervical sympathetic may become larger than its fellow even when the cervical sympathetic on the other side has not been divided.

Lodato[57] writes of the paradoxical pupillary reaction following sympathectomy and states that when chloroform is given, the pupil on the operated side dilates more considerably than on the non-operated side.

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[55] Klin. Monatsblt. f. Augenheilk., May, 1902, pp. 45, 46.

[56] Neurol. Centralbl., 20, 22, 1902.

[57] Arch. di. ottal., vol. x., 3, 4, 1902.

The dilatation must be the result of an increase in the tone of the dilatator pupillæ muscle due to the excitation, independent of the influence of the dilator nerve-fibres in their relation to the superior cervical sympathetic ganglion.

Paradoxical pupillary reflex is rare. It has been observed only in patients affected with grave lesions of the central nervous system. Bock[58] mentions two cases; both were tabetic. In one patient there were grave lesions of the sympathetic with increased redness and sweating of one side of the face. The paradoxical reaction was more marked on the side of greater redness and sweating of the face. The vision in this eye was greatly reduced. The reaction was obtained from the fellow-eye which possessed 1/6 vision but to less degree. The reaction in this case was obtained by subjecting the eyes to the influence of bright light, dilatation instead of contraction following.

Accommodative paradoxical pupillary reaction sometimes occurs in patients with grave disease of the central nervous system. Piltz mentions the rarity of true paradoxical pupillary reaction and cites but five cases, all occurring in patients with advanced disease of the nervous system.

Paradoxical pupillary reaction may be confounded with hippus, the changes in the pupil which occur in convergence and divergence, the effect of heat, and the relaxation of the pupil after forcible contraction of the orbicularis and the production of the so-called lid reflex.

More than half of the reported cases of paradoxical pupillary reaction have been reported as occurring in tabes,[59] but Uhthoff writes[60] that in all of the vast number of patients observed by him he has never seen a true case of paradoxical pupillary reaction. Cases have been observed occurring in progressive paralysis, multiple sclerosis, brain syphilis.

The Hemianopic Pupillary Reflex.[61]—Lesions of the brain situated centrally from the cerebral end of the pupillary light reflex arc, interfering with vision, do not interfere with the pupillary light reflex. A lesion situated in the internal capsule affecting the visual fibres on their way to the cortical visual area, or a lesion of the visual area, will produce blindness on one or both sides without abolishing the pupillary light reflex. This is therefore a valuable symptom in determining the location of a lesion affecting the vision. It has been suggested that the Haab reflex (page 304) may interfere with the observation of this reflex. In regard to this, Haab writes: " In hemianopsia due to cortical lesions my reflex is not a disturbing factor, as the lesion destroys the power of fixing the attention."

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[58] Graefe's Archiv, Bd. xvii., H. 2, S. 244.

[59] Muchin Money, Lancet, 1889. p. 173; Abadie and others.

[60] Graefe Saemisch, 2d, Bd. xi., T. ii. p. 240.

[61] Wernicke, Fortschrift d. Med., January 15, 1883, Bd. i., No. 2.

The relation of lesions of the cortical visual areas and the optic radiations to the pupillary light reflex may be stated as follows:

1. Lesion of the cortical visual area *on one side* gives homonymous hemianopsia of the opposite half of the visual fields, with fully preserved pupillary light reflex.
2. Lesion of the cortical visual area *on both sides* gives total blindness with fully preserved pupillary light reflex.
3. Lesion of the tractus *on one side* and the cortical visual area *on the other side* gives total blindness with pupillary light reflex preserved on the half of the retina corresponding to the side of the affected visual area.
4. Lesion of *both* tracts causes complete blindness with complete loss of pupillary light reflex.[62]

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[62] For cuts of the pupillary light reflex in hemianopsia, see Plate II facing page 290.

CHAPTER VII.

PERIPHERAL AFFECTIONS OF THE FIFTH, SEVENTH, AND CERVICAL SYMPATHETIC NERVES: OCULAR LESIONS CAUSED BY THEM; TREATMENT.

By EDWARD JACKSON, M. D.

THE FIFTH NERVE.

The differentiation between *central* and *peripheral* lesions of the fifth nerve will often depend on the location of the sensations, or the lack of sensation, experienced. Sensations referred to the whole distribution of the nerve or to widely separated portions are probably central in origin. Those confined to the region of a single branch are probably peripheral.

Anatomy.—The portions of the fifth nerve having close relations with the eye and its appendages are the ophthalmic and superior maxillary divisions, particularly the former.

Before leaving the skull *the ophthalmic division* itself divides into lachrymal, frontal, and nasal branches, which enter the orbit through the sphenoidal fissure. The lachrymal branch, entering the orbit towards the temporal end of the fissure, passes to the region of the lachrymal gland, to which it supplies several filaments; and is distributed to the conjunctiva and portions of the skin indicated in Fig. 1.* The frontal branch, entering through the upper middle portion of the sphenoidal fissure, passes forward near the roof of the orbit to the supra-orbital foramen or notch, through which a portion emerges as the supra-orbital branch, the supratrochlear having been previously given off to emerge from the upper inner angle of the orbit. The nasal branch, entering the orbit slightly to the nasal side of the frontal branch, passes along the nasal wall of the orbit, enters the ethmoid through the anterior ethmoidal foramen, and is distributed chiefly to the nose (See Page 15).

The superior maxillary division of the nerve, passing through the foramen rotundum across the sphenomaxillary fossa and through the infra-orbital canal emerges from the infra-orbital foramen. Its temporal and malar branches, given off in the sphenomaxillary fossa, pass through the malar bone; and the palpebral and nasal branches, given off after it emerges from the foramen, are distributed to the lids, conjunctiva, and region of the lachrymal sac.

The general distribution of the branches of the fifth nerve to the skin of the head and face is shown in Fig. 1.

The most important anastomoses of the trigeminus are those with the sympathetic nerves, especially its connection with the ciliary ganglion. It should be borne in mind that beside the superficial distribution indicated in Fig. 1 the fifth is also the nerve of general sensation for the anterior part of the dura mater, and the mucous membrane of the ear, nose, throat, accessory sinuses, the mouth, and the upper portion of the pharynx.

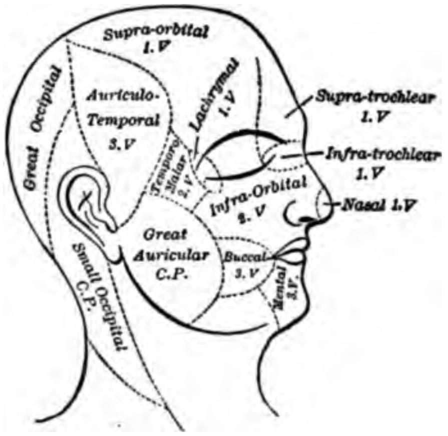

FIG. 1.—Normal distribution of the fifth nerve to the face. *1.* V—ophthalmic division; *2.* V—superior maxillary; *3.* V—inferior maxillary. The names on the different areas indicate the branches supplying them. (Flower.)

Physiology.—Besides serving to transmit the impressions of touch, pressure, and temperature, and to excite pain, the fifth nerve is concerned in very important reflexes. Hyperæmia in response to corneal irritation may involve the vessels of the optic disk and retina as well as those of the pericorneal zone. Contraction of the pupil in response to such irritation is often very striking. The increase of the lachrymal secretion may be a reflex response to irritation of any portion of the mucous membrane supplied by this nerve. Its fibres may also carry important efferent impulses governing secretion, or the nutrition of the various tissues (trophic).

Anæsthesia.—In rare cases absence of sensibility over parts or the whole of the distribution of the fifth nerve appears to be due to congenital defect. More frequently it arises from lesions due to pressure, inflammation, or toxic disturbance of the nerve trunk or its branches. Chief among these toxic influences are malaria, lead, and auto-intoxication of intestinal origin. The location of the anæsthesia will be determined by the nerve distribution. Thus, small areas indicate that the course of the disturbance is acting upon correspondingly few peripheral filaments. Anæsthesia involving the whole distribution of an important branch, such as the supraorbital, indicates that this nerve is affected before breaking up into its terminal filaments. When the anæsthetic region involves the distribution of all the branches of one of the great divisions of the nerve—the ophthalmic or the infraorbital—the cause must act close to where such divisions emerge from the skull. The anæsthesias of more peripheral origin, and therefore involving smaller areas, are very much more likely to be overlooked. This may be the reason that the extensive anæsthesias of intracranial origin appear to be the more common.

Corneal anæsthesia is of especial interest. The sensibility of the cornea is commonly tested by touching it with a thread or a few fibres of absorbent cotton rolled into a point. For purposes of comparison, the sound cornea may be similarly tested in alternation with the cornea suspected of anæsthesia or hyperæsthesia. The sensitiveness is to be judged by the apparent reaction produced, as well as by the patient's statements.

The relative ease with which anæsthesia of the cornea is produced by the use of cocaine, holocaine, and other local anæsthetics seems to indicate that there is an especial liability of the corneal nerves to such functional impairment. In malarial disease anæsthesia of the cornea may be demonstrated when no other evidence of trigeminal anæsthesia can be discovered. The writer has observed it confined to a certain segment of the cornea, the sensibility of other parts being normal. The corneal anæsthesia of glaucoma is ascribed to pressure on the nerve-trunks as they pass forward within the sclera. It is proportioned to the increase of the intra-ocular tension, and to the rapidity of that increase.

Paræsthesia.—Perverted, abnormal, and usually disagreeable sensations—like burning, pricking, tingling, numbness, or fulness of the parts supplied by the fifth nerve—are more frequently recorded as due to central causes. But they may arise from abnormal states, as neuritis of the peripheral branches. After injuries—particularly those causing bruising of the soft tissues, or fracture of the bones about the orbit—such sensations may persist for many months or even for several years. Gradually they become less constant and less annoying until they entirely disappear. After some ocular inflammations, especially inflammations of the iris and cornea, sensations of the kind may be complained of for considerable periods. Some of the discomforts which persist after the removal of the eye-strain that had been attended by chronic congestion, may be of this character.

In overcoming these disagreeable sensations, time is the most important factor, but something may be done by avoiding, so far as possible, any source of irritation or strain, or any observed exciting cause.

Neuralgia and Neuritis.—Neuralgic pain of peripheral origin is probably generally due to neuritis. At least, in practice we are unable to distinguish between pain which is due to neuritis and that which is not. When such pain is confined to a small area of the skin distribution of the fifth nerve, its character may be readily recognized. But when it is referred to the eyeball itself, its significance becomes more difficult to estimate. The latter will be considered under the next heading. Neuralgic pain attended with facial spasm (prosopalgia or tic douloureux) is discussed under facial spasm.

Ocular Pain.—It is very important, but often difficult, to determine the exact character of pain referred to the eye. The two most important varieties are the pain such as is produced by irritation of the conjunctival or corneal nerve-endings, and such as would arise from deeper lesions. The former is described as scratching, smarting, or the feeling of a foreign body; the latter rather as aching. Burning and itching may arise from either set of causes, but these words are more commonly used with reference to pain of superficial origin. Boring, shooting, or stinging pains are usually of deep origin, although the latter terms may be applied to sensations better referred to as scratching.

The exact location to which pain is referred may be of important significance but it cannot be wholly relied on. By the scratching felt in a certain position, the location of a minute foreign body or superficial lesion may be ascertained. But with regard to pain of deeper origin, its referred location is likely to prove misleading. The pain of iritis or glaucoma, if severe, will always be felt more or less in the brow or cheek. Often it is complained of chiefly or entirely in these regions. The mistaking of these diseases for trigeminal neuralgia is extremely common among practitioners whose attention is not especially fixed upon the eye. Pain referred to the distribution of the ophthalmic or superior maxillary divisions of the fifth nerve should always lead to a careful examination of the eye-ball; and pain of ocular origin may be even more remotely referred.

The persistence of paræsthesia after injury has been referred to. Pain may be equally persistent. Even though no organic disease may be discoverable, pain may continue as a sort of morbid habit. This is noticed especially after the relief of eye-strain that has long caused headache. But in such cases the suffering gradually diminishes. Sometimes after an operation on the eyeball or after accidental injury or severe inflammation, pain out of all proportion to the severity of other symptoms may persist for a long time. In these cases it might reasonably be supposed that a neuritis of the nerve-endings of the fifth nerve had arisen; and treatment directed in accordance with this supposition gives, after the necessary lapse of time, satisfactory results.

Neurotic Œdema.—Localized œdema of the conjunctiva or lids, or both, apparently dependent on nerve influence, is not very rare. Sometimes it is accompanied by vascular engorgement of the part. But in many cases this is not noticeable. The acute attack may be ushered in by burning, stinging, or itching of the parts, followed by very rapid swelling, which may cause complete closure of the eye. Usually within twelve to forty-eight hours the swelling begins to diminish, and in three or four days the parts are restored to their normal appearance. Commonly but one eye is affected, but the swelling may extend from one side to the other, or both may be originally attacked. Often there is similar acute œdema of other parts. But the ocular symptoms may be the only ones complained of.

Some of these attacks closely resemble urticaria in their origin and nature. They may be provoked by use of certain articles of diet, as fish or shell-fish, or the drinking of alcoholic beverages. In a few persons large doses of potassium iodide will produce enormous conjunctival swelling of this character.

Chronic œdema of the lids, liable to increase from errors of diet, exposure to wind or dust, use of the eyes, or loss of sleep, is sometimes a very annoying and obstinate condition. It may continue to give trouble for many years. The treatment includes protection of these parts from local irritation, and careful avoidance of the exciting cause, when this can be discovered.

Trophic Disorders.—It is uncertain whether trophic disorders in the region of the fifth-nerve distribution are often of extracranial origin. But, as their peripheral manifestations are well known and the causative lesions unknown or extremely obscure, it is convenient to consider them in this connection.

Facial hemiatrophy involves to some extent the lids and the bones of the orbit, but produces no evident alterations in the eyeball itself. Unilateral hypertrophy of the face may involve the same parts. Whitening or falling of the lashes or hairs of the eyebrow on one side, or a portion of them, is ascribed to morbid nerve influence. Disorders of secretion, hyperidrosis, or chromidrosis, localized erythema, itching, and perhaps even gangrene, may have a similar origin.

Neuropathic Keratitis.—Magendie observed that section of the trigeminus in rabbits was followed by inflammation and destruction of the cornea. He assumed that this depended upon the loss of a trophic influence necessary to sustain the normal corneal nutrition. Snellen suggested that lack of sensibility in the cornea permitted injuries to occur to it; and foreign bodies to remain in contact with it, by suspending reflex movements of the lids and lachrymation; and it was shown that after section of the trigeminus, careful protection of the eye prevented keratitis. Keratitis can be prevented by fully protecting the eye for a few weeks after operative removal or destruction of the Gasserian ganglion. Claude Bernard called attention to the share which vasomotor changes might have in producing this form of corneal inflammation. Turner and Ferrier, by experimental divisions of the fifth nerve, its roots and branches, with protection of the eye from injuries, reached the conclusion that keratitis from injury or disease of the trigeminus was the result of an irritation rather than a paralysis.

Many recent writers, apparently from an incomplete survey of the subject, have accepted the explanation of Snellen, that it was a keratitis of traumatic origin, which might be rendered grave by intercurrent infection. But, as pointed out by Harlan,[1] such an explanation does not meet all the known facts. The clinical appearance and course of the disease differ radically from those of an ordinary traumatic infective keratitis. Harlan reported a case in which the cornea remained clear under prolonged exposure due to facial paralysis, and subsequently sloughed when it was protected by paralysis of the elevator of the upper lid. In a case reported by Norris the sloughing of the cornea occurred in spite of careful protection against injury. It must also be borne in mind that neuropathic keratitis occurs in connection with trigeminal disease without anæsthesia of the cornea, and that corneal anæsthesia due to glaucoma is not attended with any such inflammation.

In the present state of our knowledge a rational view seems to be, that disease or injury involving the trunk or ganglion of the fifth nerve causes or predisposes to corneal disease, partly by an obscure irritative influence and partly by favoring traumatism and infection. But the different hypotheses above mentioned should all be borne in mind, since trophic influence, injury, vasomotor disturbances, and central irritation may all be factors in the case and may require to be met by different therapeutic influences.

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[1] Transactions of the American Ophthalmological Society, 1897.

The clinical course of the disease may be that of a general softening or sloughing of the cornea. More commonly, as the writer has seen it, it begins with clouding of the central portion of the cornea. This clouding impairs vision. It is accompanied by some general hyperæmia of the eyeball, but with comparatively little inflammatory pain or reaction. Lachrymation is not increased, but the corneal surface is not especially dry. The sensibility of the cornea is usually markedly diminished. But this may not be the case. Ulceration is liable to occur first in the central portion of the cornea.

The ulcer may become infected, hypopion form, and the cornea slough. In a fair proportion of cases the process does not go on to perforation; but the cornea heals with permanent opacity. Central vision is thus greatly impaired or destroyed, but the eye may still preserve a useful peripheral field. The corneal disease may be attended from an early stage with evidences of iritis, including adhesions, swelling, and discoloration of the lids.

Some writers have applied the description of keratitis due to exposure and desiccation of the cornea—*lagophthalmic keratitis*—to this disease. The two conditions, however, differ markedly in important clinical features. Keratitis from exposure begins with dryness, ulceration, and infection in the lower part of the cornea, while in true neuropathic keratitis it is the central region of the cornea that is first and most severely affected. Exposure keratitis has the appearance and runs the course of an infective ulcer. Neuropathic keratitis shows a deep opacity of the central part of the cornea preceding or without ulceration.

Treatment.—The local treatment of neuropathic keratitis includes careful protection of the cornea by union of the lids or the occlusive bandage, the cautious application of hot stupes or dry heat, careful cleansing of the eye with physiologic salt solution or very weak antiseptic solutions, and the instillation of atropine. The prognosis, however, must depend largely upon the nerve element in the case. If this can be improved by general regimen, tonics, or the very cautious application of electricity, such measures must not be neglected.

Herpes Zoster.—Numerous cases of corneal leucoma, associated with scars on the face and scalp in the area of distribution of the ophthalmic division of the trigeminus, with the history that this corneal disease occurred with an attack of what was regarded as erysipelas, called the attention of Jonathan Hutchinson, Sr., to the importance of the eye lesions of herpes zoster affecting this region. An acute clinician ought to be able at any stage to recognize that he has to deal with zoster rather than erysipelas; and yet the diagnosis is easier after the case has run its course than it is in the early stages.

Herpes, unlike erysipelas, is preceded by severe burning, neuralgic pain in the affected part, and is not preceded by a chill, high fever, marked headache and other evidences of severe general sickness. The eruption of herpes zoster begins with isolated spots, upon which vesicles develop, instead of having the redness and swelling general from the start. After the eruption begins to appear, there is no extension of the region affected, and there is but little involvement of the lymphatic glands.

The disease is unilateral, the eruption being sharply limited near, but often not exactly at, the median line. The distribution corresponds to the more or less variable areas of nerve distribution. The skin of the whole affected region becomes inflamed, causing severe swelling of the lids and the appearance which gives rise to the diagnosis of erysipelas. The eruption runs its course in from one to three weeks, each vesicle giving rise to a scab which falls off leaving a permanent depression. The neuralgic pain may disappear with a full development of the eruption, but often it persists, especially in elderly people, after all inflammatory symptoms have disappeared, sometimes causing great suffering for many weeks or months.

The ocular lesions attending ophthalmic herpes are quite varied. Wilbrand and Saenger have brought together cases of involvement of the iris, ciliary body, chorioid, retina, and optic nerve, increase and diminution of intra-ocular tension, and paralysis of the internal and external muscles of the eye. In a few cases it has been followed by paralysis of the facial nerve. These complications appear to present the usual characters of inflammation, degeneration, or paralysis of these respective parts.

The common, dangerous, and characteristic ocular lesion is presented by the cornea. With the development of the skin eruption vesicles may appear on the conjunctiva and cornea, giving rise in the latter situation to corneal ulceration. The corneal ulcer may extend and perforate. But without perforation and even without ulceration, while the eruption is at its height, a dense opacity may develop in the depth of the cornea, leaving a permanent leucoma or dense nebula. The corneal inflammation is apt to last much longer than the skin eruption with which it begins; and such clearing of the opacity as does occur takes place very slowly, and may not begin until after the persistence of the neuralgic pain has ceased to give serious annoyance. The cornea will often be found quite anæsthetic, the anæsthesia continuing through the period of pain and maximum opacity. In many respects the corneal disease has the characteristics of neuropathic keratitis.

Causes.—The pathology of herpes zoster is not fully worked out. But the ocular and skin lesions seem to depend upon inflammatory changes, hemorrhage, and subsequent degeneration in the Gasserian ganglion. The inflammation may extend to the nerve-trunk, and the degenerative changes through the roots of the nerve into the central nervous system. Head and Campbell[1] believe that herpes zoster is a specific disease with a definite period of incubation, and occurring in epidemics. More generally it has been ascribed to cold or to some toxic influence. Arsenic and exposure to charcoal fumes have been regarded as especially liable to cause it.

Treatment.—The local treatment should be the same as for neuropathic keratitis, protective and non-irritative (see page 848). When all pain has disappeared and the sensibility of the cornea has been largely restored the usual remedies for corneal opacity may be resorted to. But the permanent opacity is likely to be out of proportion to the loss of corneal tissue.

Prognosis.—The prognosis varies with the severity of the attack, the presence of ulceration, deeper opacity of the cornea, and the occurrence of iritis, or involvement of other deeper structures of the eye. In general the danger of a prolonged or permanent disability of the eye increases with the age of the patient. It has been noted that the cornea is more likely to suffer in those cases in which vesicles appear on the side of the nose. But severe corneal lesions may occur when the skin of the nose is not at all involved, and the cornea may escape, although well-marked vesicles or scars are found upon the nose. In all cases the prognosis should be guarded, especially as to the time of recovery. Some eyes remain irritable and painful many months and years after the attack, and there is no way of judging in the early stages which cases will run such a course.

Other Forms of Herpes.—It must not be forgotten that other forms of herpes occur in and about the eye. In rare cases the ordinary febrile herpes has been observed occurring on the lids as it generally occurs upon the lips. Herpes of the cornea occurs in connection with various febrile diseases, especially affecting the respiratory tract. Symptoms of severe corneal irritation attend the development of the vesicles, and their rupture leaves a number of superficial ulcers. But these ulcers commonly heal without leaving any permanent impairment of vision. Phlyctenular disease of the conjunctiva and cornea also exhibits many of the characteristics of an herpetic eruption. There can be little doubt that the peripheral branches of the fifth nerve perform an essential part in the production of all these lesions. In febrile herpes the disturbance may be due to poisoning or impaired nutrition of the nerve. In phlyctenular disease it has been ascribed to the local action of various bacterial toxines.

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[1] Brain, 1900, No. 91.

Recurring Erosion of the Cornea.—After a superficial injury, as a finger-nail scratch of the cornea, attacks of severe pain with or without loss of epithelium may recur for months or even years. Injury to the peripheral nerve-endings of the cornea is probably an essential factor in producing this condition.

Dendritic ulcer of the cornea, as has been pointed out by Charles,[1] probably owes its peculiar character to lesions affecting the terminal filaments of the corneal nerves between Bowman's membrane and the epithelium.

Supposed Reflex Blindness and Palsies.—From the time of Hippocrates it has been known that various injuries in the region supplied by the fifth nerve were liable to be followed by blindness or by paralysis of one or more of the ocular muscles. It is now known that in many of these cases the blindness or paralysis is due to direct injury to the optic nerve or to the motor nerve supplying the affected muscles. Wounds about the orbit often penetrate more deeply than is suspected, and hemorrhage or thrombosis may carry their effects still deeper. Post-mortem examination has demonstrated that of fatal cases of fracture of the cranium from blows upon the region supplied by the fifth nerve the fracture involved the orbit in 68 per cent., and in 51 per cent. extended into the optic foramen.

Nevertheless, the view that optic nerve atrophy and paralysis of the motor nerves of the eye might be produced by trophic influences set up by injury to the peripheral branches of the fifth nerve has been accepted by many excellent observers, even in recent years. Hubbell,[2] who has recently supported this hypothesis, cites a long list of famous clinical observers who are inclined to accept it. To the writer it seems more reasonable to suppose that all cases of the class are caused in the same or closely similar manner in which we know that a large proportion are caused, than to have recourse to an hypothèsis so indefinite and unsupported as that of a harmful trophic influence exerted by the fifth nerve.

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[1] American Journal of Ophthalmology, April, 1904.

[2] Transactions of the Section on Ophthalmology of the A. M. A., 1904.

Photophobia.—Although the term photophobia (fear of light) might be expected to indicate only symptoms arising from the retina and optic nerve, it is applied to phenomena more generally connected with irritations acting upon the peripheral branches of the fifth nerve. It is easy to see how light falling upon an eye with an inflamed iris may cause disturbance of the nerves of general sensation distributed to the iris by the reaction it provokes causing movements in the inflamed tissue. But in some of the most marked cases of photophobia it is evident that light has little or nothing to do with the production of the symptoms. The child with phlyctenular keratitis buries its head in the pillow as strenuously and persistently at night or in the darkened room as in full daylight. And its fear of light is removed by measures that diminish the hypersensitiveness of the fifth-nerve endings, but which have no appreciable effect upon the retina and optic nerve.

There is a form of discomfort about the eyes, and aching often becoming a general headache, which seems directly connected with exposure to light. This may be experienced by most persons who are much indoors with feeble illumination. When they go directly into strong sunlight, the pain may arise instantly, and may have the character of a sharp cramp, probably connected with excessive contraction of the sphincter of the pupil. The contrast between bright lights and comparative darkness, produced by looking towards sources of artificial illumination at night, is quite unpleasant to most people. Excessive liability to similar unpleasant sensations is noted in patients suffering from acute or chronic wasting disease, attended with toxæmia, especially after typhoid fever and in the course of general tuberculosis. Photophobia of the kinds above mentioned may have little connection with the fifth nerve, and that only through the nerve-centres.

But the photophobia which attends the presence of a foreign body in the eye, or inflammatory irritation of the conjunctiva, cornea or iris, is clearly a fifth-nerve symptom. It is associated with no characteristic anatomical changes in the nerve. Indeed, the production of this form of pain can hardly be regarded as an abnormality of the nerve function, since it seems necessary to secure rest and protection or relief from the irritation that causes the photophobia. In this form of photophobia there is a tendency to spasmodic closure of the lids, excessive lachrymation, and general hyperæmia of the eyeball. In the more severe cases the face may be continuously buried in a pillow, cushion, or handkerchief, or the eyes exposed to the air and light only with great difficulty, and for a short time.

Treatment.—Photophobia which is rarely connected with exposure to light should be met by measures that will lessen the general erethism of the nervous system, and by avoiding contrasts of illumination, either successive, by going from darkness suddenly to light, or simultaneous, as by facing sources of artificial light at night, or the narrow streaks of light about a shaded window. But it is important not to have the patient give way too much to the desire to avoid diffuse daylight. If dark glasses are worn they should be resorted to only at times of exposure to the brightest light, so as to lessen the contrast. They should not be used continuously. Habitual exposure to moderate light is the physiologic and most efficient method of keeping down the hypersensitiveness of the retina.

Photophobia dependent on corneal irritation demands chiefly the removal of the irritation. The avoidance of exposure to light and air may, however, tend to increase the photophobia. Often the dressings worn upon the eye aggravate and continue the symptom when it would otherwise have subsided. Photophobia is usually an indication for removal of bandages and avoidance of anything that will keep up the heat and moisture of the parts. It is often wise to substitute dark glasses for a close dressing, simply because they permit the free access of air, and continuous cooling of the parts by evaporation. Strongly astringent applications, as silver nitrate, boroglyceride, etc., do good by removing the hypersensitiveness of the nerve-endings.

In photophobia attended by lachrymation small fissures in the skin are apt to appear about the outer canthus (rhagades). The treatment of these, as by touching with a strong solution of silver nitrate, will greatly lessen the photophobia. The local application of cocaine or holocaine within the conjunctival sac temporarily relieves photophobia. But with the passing of the anæsthetic influence the symptom returns with renewed force, and frequent reapplications of cocaine to keep up the anæsthetic influence are extremely dangerous.

In corneal disease the influence of atropine upon the nerve-endings in the cornea gives a decided and much more permanent relief, and usually proves beneficial also for the condition causing the photophobia. Where photophobia is attended with spasmodic closure of the lids and does not yield to other measures, it will usually be greatly lessened by canthotomy or canthoplasty. The old plan of suddenly plunging the head into cold water is efficient, but not often practical.

alone but not the other. The latter is commonly the case where one eye has distinctly better vision than its fellow. It is better to have the patient try to close both eyes at once or to elevate both brows as nearly alike as possible. One may also detect the lessened power of the muscles by offering resistance to their action, as by resisting closure of the lids. This may be made a rough test of recovery.

While in paralysis of central origin the oculofacial muscles are usually but slightly involved or may quite escape, it is possible to have a peripheral paralysis confined to these muscles. Thus Mills mentions a case in which from a stab in front of the ear the frontalis was paralyzed and the orbicularis palpebrarum was weakened, although the other facial muscles were unaffected. In another case, following a fall on the temple, the loss of power was confined to the frontalis (page 439).

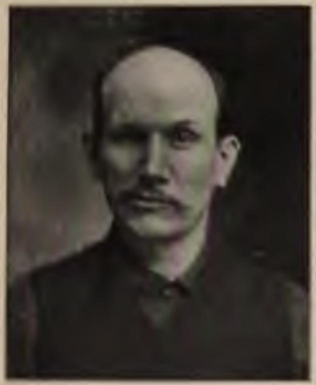

FIG. 5.—Contracture of the orbicularis palpebrarum and of other muscles of the right side of the face in facial paralysis of central origin. (Case of Dr. Spiller.)

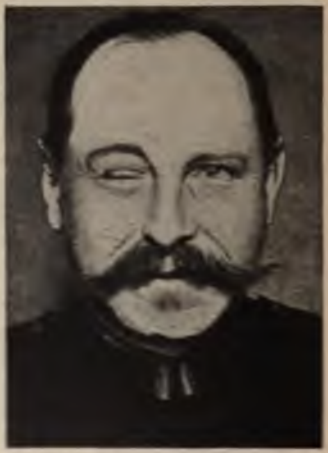

FIG. 6.—Total peripheral facial paralysis of right side. Secondary contracture. Involuntary closure of eye and elevation of brow in laughing. (Wilbrand and Saenger.)

Paralysis of the frontalis destroys the power of elevating the eyebrow. This leaves the brow drooping on the affected side, narrowing somewhat the upper part of the field of vision. Paralysis of the orbicularis prevents closure of the lids. The consequences of exposure of the eye will be discussed under lagophthalmos. It also permits the lower lid to hang away from the eyeball, interfering with the lachrymal function. (See LACHRYMATION.) After three or four months secondary contractures may occur in the paralyzed muscles. These are less frequently exhibited in the oculofacial muscles than in those acting on the mouth. But they may obscure the diagnosis, and even give the impression that the paralysis affects the opposite side (Fig. 5).

The contracture of the orbicularis causes narrowing of the palpebral opening and wrinkling of the neighboring skin. The contracture of the frontalis causes an undue elevation of the eyebrow of the affected side. They are increased during voluntary action of the affected muscles, as in laughing. The effects of such contracture are well shown in Fig. 6.

Etiology.—A large proportion of cases give a clear history of recent special exposure to cold, which seems to act by setting up a neuritis or perineuritis. Less frequently the neuritis seems to be of toxic origin. Syphilis, rheumatism, diabetes, gout, leukemia, diphtheria, and other specific infections are credited with causing it. It has also been ascribed to an acute specific infectious process. A considerable number of cases are secondary to aural disease, particularly suppuration of the middle ear, and local injury to the nerve or its branches may cause it. It may occur as a part of a polyneuritis. Shumway[1] has collected eight cases in which it has been accompanied by optic neuritis and subsequent optic atrophy. In two cases he has seen it followed by marked flattening of the face on the affected side and ptosis.

Treatment.—This must first combat the cause. In neuritis due to cold it is important that active treatment should begin promptly. A general purgative, leeches below the ear, salicylates and later iodides are to be tried. Counterirritation by a blister or by milder irritants may prove useful. At a later stage strychnia may be given. Electricity is to be used with caution, and its application immediately suspended if there arises evidence of secondary contracture (page 885).

Prognosis.—The prognosis depends upon the cause and, with neuritis due to cold, somewhat on the promptness with which effective treatment is begun. Probably half of all cases make a fair recovery, but a considerable proportion show permanent loss of power in the affected muscles, and in not a few the paralysis continues complete. The rapid and complete loss of faradic irritability is unfavorable, and complete recovery cannot be expected where secondary contractures have occurred. Long duration of the paralysis is also a ground for an unfavorable prognosis, although cases may recover after several months.

Facial Spasm.—This is more frequently of cerebral or nuclear than of peripheral origin. But it may be caused by a gross irritative lesion in any part of the nerve-trunk or its branches. Meige[2] classes as "spasms" those motor reactions arising from actual irritation, and as "tics" those disorders which have a psychomotor origin, and which are therefore not discussed in this chapter. Certain forms of spasm are discussed in connection with the palpebral opening. We are here concerned with spasm of the frontalis, the corrugator supercilii and the orbicularis palpebrarum muscles, and only the last, blepharospasm, is of much practical importance.

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[1] Trans. Sec. on Ophthalmology, A. M. A., 1904.

[2] Annales d'Oculistique, March, 1903.

Blepharospasm. — Blepharospasm varies from slight fibrillary twitchings of the orbicularis muscle, to painful contraction of the muscle, which resists strong mechanical effort to separate the lids, and entails periods of practical blindness. The fibrillary twitchings may be observed shortly after the instillation of eserine. Many persons experience them after loss of sleep or moderate overexertion of the eyes. They are often quite annoying to the patient, although scarcely perceptible to anyone else. They have no grave significance, and can be relieved by sleep, removal of any local irritation or of eye-strain. But they are liable to recur in persons predisposed to them.

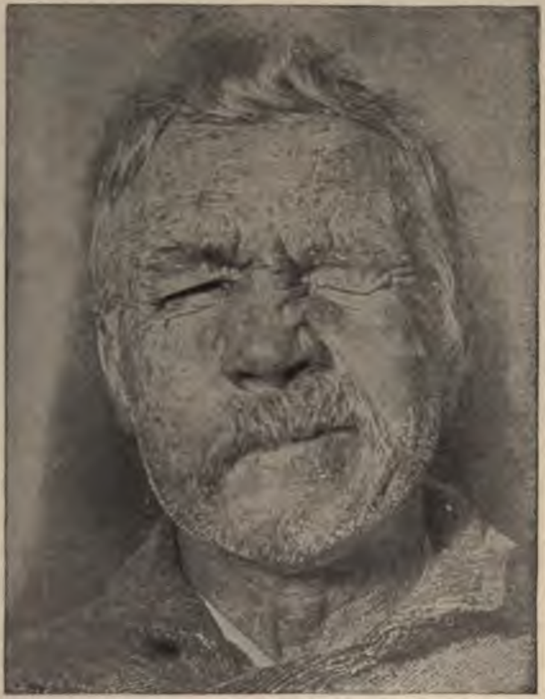

FIG. 7.—Case of tic douloureux: appearance of patient's face during severe paroxysm. (Mills).

Excessively frequent, somewhat prolonged acts of winking are often noticed in children. They call for careful search for some local irritation or eye-strain. Older persons, who have usually suffered from prolonged conjunctival irritation, acquire a habit of exaggerated winking, a rather prolonged, forcible closure of the lids. This habit becomes a serious complication when certain operations, especially cataract extraction, are required.

More acute and often uncontrollable blepharospasm is a symptom of conjunctival, and especially of corneal, irritation. It is usually associated with photophobia and excessive lachrymation. This is particularly marked as a symptom of phlyctenular disease. The pulling of a tooth will cause a sudden reflex spasm of all the muscles of that side of the face. Forcible uncontrollable spasm of the lids occurs in tic douloureux or prosopalgia (Fig. 7). Hysterical blepharospasm is discussed in the chapter on hysteria (page 651). In a few cases attacks of spasmodic closure of the eyes without discoverable cause occur with gradually increasing frequency and prolongation of the attacks. Mills suggests that these are due to nuclear instability,—a manifestation of chronic degenerative changes.

Diagnosis.—All forms of blepharospasm tend to cause narrowing or closure of the palpebral opening, and wrinkling of the skin of the lids and adjoining parts. Ptosis causes narrowing of the palpebral opening, but is rather attended with smoothness of the lids from relaxation or distention of the parts. But, besides recognizing the condition present as blepharospasm, the diagnosis must include the determination of its cause, if possible. Careful search should be made for a source of irritation, as inflammation or the presence of a foreign body. Beginning with the cornea and conjunctiva this search must extend to the regions supplied by other branches of the fifth nerve, including investigation of possible disease of the maxillary, nasal, and other adjoining sinuses, and of the teeth.

Treatment.—The treatment of blepharospasm is first, and usually wholly, the removal of its cause. This must often include the correction of ametropia or other possible cause of eye-strain, as well as the removal of mechanical or inflammatory irritation. In a few chronic cases in which the spasm causes severe pain or temporary disability by blindness, it is right to resort to stretching, division, or excision of the nerve-branches concerned. These measures will give relief for the time, sometimes for many months, but their effect is not often permanent (p. 894).

Prognosis.—This is favorable in proportion as the spasm is due to an evident and remediable irritation. Cases not so caused, and especially those due to chronic degenerative changes, are well-nigh hopeless. Certain tics of central or psychomotor origin have a better prognosis, being amenable to educative treatment.

Spasmodic Entropion.—The tendency to exaggerated winking produced by any irritation in the conjunctival sac, with the chronic inflammatory changes that produce it, are the causes of spasmodic entropion. Entropion caused by relaxation or swelling of the skin of the lids is often classed with this. But the true spasmodic form depends on the action of the orbicularis. When this muscle is strongly contracted, the palpebral opening is narrowed, and the skin around is dragged in towards it. When this dragging is kept up, or repeated at short intervals over a considerable length of time, the skin at the lid margin becomes more or less pushed over the edge of the cartilage toward the inner side of the lid, carrying with it the lashes, which then rub against the eyeball, and still further increase the irritation and tendency to spasmodic contraction of the orbicularis. Entropion most frequently occurs in the course of trachoma, in which there are, with conjunctival irritation, cicatricial changes in the lids leading to incurving of the tarsus and organic entropion. In these cases it is hard to separate the spasmodic from the cicatricial causes. But the orbicularis spasm may be an important factor in the development of organic entropion.

Treatment.—This must include, first, the removal of the irritation which is provoking the orbicularis to spasmodic contraction, as the treatment of the trachoma. In some cases the habit of spasmodic closure of the lids is itself an important cause of irritation, and canthotomy or canthoplasty, preventing the excessive pressure of the lids upon the eyeball, will be followed by great improvement. In severe chronic cases, however, an entropion operation is indicated, especially that of Hotz, which fastens the skin of the lid securely to the upper margin of the tarsus.

THE SYMPATHETIC NERVE.

Anatomy.—The sympathetic nerve supply of the eye and its appendages comes through the superior cervical ganglion of the sympathetic, which Langley calls "a relay station for the sympathetic nerve supply for the whole of the head." From this ganglion branches form plexuses which accompany all the arteries distributed to the orbit and surrounding regions, and other branches, mostly from the larger plexuses, join the cranial nerves. The third receives such a branch at its point of division; the fourth as it lies in the wall of the cavernous sinus; the fifth in the Gasserian ganglion; the sixth where it lies in close apposition with the carotid plexus; the seventh at the stylomastoid foramen.

In the orbit is situated the *ciliary ganglion.* This is about 2 millimetres long and 1 millimetre thick. It lies slightly to the temporal side, and slightly lower than the optic nerve, 8 to 11 millimetres from the optic foramen, and 14 to 17 millimetres from the entrance of the optic nerve through the sclera. Its roots are derived from the fifth nerve, usually from a branch of the nasal just after entering the orbit, a filament of the carotid plexus, and a filament from the oculomotor, usually from the branch supplying the inferior oblique. It gives off the short ciliary nerves, which enter the posterior part of the sclera, and run forward between it and the chorioid to the ciliary muscle and iris. We thus have the sympathetic reaching all parts of the eye and its appendages, and a special mechanism supplying the iris and ciliary body, the function of which is discussed in Chapter VII. on " The Intra-ocular Muscles."

Vasomotor Disturbances.—The most general and best understood function of the sympathetic nerve is its vasomotor function. Unilateral flushing of the face and neck occurs, and may be attended with noticeable hyperæmia of the conjunctiva and narrowing of the palpebral opening. Such an attack may last but a few minutes or it may continue for hours. A more permanent impairment of this function may occur. Following influenza, the writer has seen hyperæmia of one eye, especially of the conjunctiva, with narrowing of the palpebral opening and slight tonic ptosis. Cases of this kind have not received the attention they deserve. It is very easy to confuse such a condition with unilateral inflammation of the conjunctiva and partial closure of the lids on account of irritation. But instead of active local treatment these are to be benefited chiefly by improving the general condition. Pulsating exophthalmos not due to gross lesions of the orbital vessels may be regarded as a vasomotor disorder.

The connection of the general blood-pressure with the intra-ocular tension has been the subject of various theoretic discussions and physiologic experiments. The conclusions drawn from the latest of these by Parsons and Snowball [1] are that the intra-ocular tension responds passively to all variations in the general blood-pressure, whether these be produced by vasomotor actions or by such mechanical means as the tying of a large artery or by the inhibition of the heart's action. The increase of intra-ocular tension caused by stimulation of the peripheral portion of the cervical sympathetic they ascribe, however, not to vasomotor action but to contraction of the unstriped muscles of the orbit.

Influence of Unstriated Muscles.—Stimulation of the sympathetic produces contraction of the unstriated muscle-cells with which it is connected. The intra-ocular muscles and their action are discussed in the preceding chapter. The principal extra-ocular muscles of this character are those acting upon the upper and lower lids; and the fibres stretched across the sphenoidal and sphenomaxillary fissures. These latter are better developed in some of the lower animals; and it is doubtful if they have much practical importance in the human orbit.

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[1] Royal London Ophthal. Hosp. Reports, vol. xv. Part 3, p. 275.

Contraction of Mueller's muscles connected with the lids tends to cause retraction of the lids. Their relaxation produces narrowing of the palpebral opening. Contraction of fibres stretching across the back of the orbit has been suggested as the explanation of the protrusion of the eyes produced by stimulation of the sympathetic, and relaxation of these fibres has been supposed to account for exophthalmos. But it is doubtful whether the protrusion and sinking, produced by stimulation or paralysis of the sympathetic in man, are not merely apparent rather than real, the appearance being produced by the widening or narrowing of the palpebral opening. The detailed effects produced by these muscles are discussed in connection with alterations of the lids and the palpebral opening.

Secretion. One sided sweating with flushing of the face occurs from defective sympathetic innervation. In these cases the eyelids show slight increase of secretion. The influence of the sympathetic upon the lachrymal secretion is discussed under that heading. What share its influence on secretion has in affecting the intra-ocular tension will be considered under the next heading.

Intra-ocular Tension. Softening of the eyeball after section of the cervical sympathetic was one of the earliest observations regarding [illegible] of this nerve. Increase of intra-ocular tension by stimulation [illegible] of the nerve is another common observation. [illegible] intra-ocular tension are produced is still [illegible] influences pro- [illegible] of the eye. Adamük [illegible] especially to the [illegible] concluded that the [illegible] suggested that [illegible] increased tension. [illegible] of the superior [illegible] same [illegible] normal [illegible] as a [illegible] the changes of tension produced by disturbance of the cervical sympathetic were but brief. De Schweinitz [1] concludes that the fall of the intra ocular tension produced by section of the sympathetic or extirpation of the sympathetic ganglion probably depends on vascular or perhaps muscular changes.

It is quite possible that the sympathetic exerts, apart from its vasomotor function, a direct control over secretion within the eye. But no one has yet been ingenious enough to devise appropriate experiments or to so marshal clinical facts as to clearly demonstrate this condition.

Within the last ten years excision of the superior cervical ganglion of the sympathetic has been done many times for exophthalmic goitre, epilepsy, and glaucoma. From the clinical observations on these cases and those in which the sympathetic has been paralyzed, as by pressure from enlarged cervical glands, it seems clear that loss of function of the cervical sympathetic is followed by diminished intra-ocular tension. In healthy eyes this diminution seems to be brief and unimportant. In glaucomatous eyes it may be more prolonged.

Wilder [2] concludes that "sympathectomy for glaucoma is not an operation to be condemned too hastily. . . . The statistics up to date seem to indicate that the simple chronic form is the one most suited for it, next to the hemorrhagic form, if that can be determined." What is yet known with reference to the influence of the sympathetic upon intra-ocular tension does not justify neglect of older established methods of controlling this condition. But it does justify resort to sympathectomy as an adjunct to other treatment or when such treatment has proven insufficient.

Excision of the ciliary ganglion practised by Rohmer [3] is a difficult operation on account of the smallness and position of the ganglion; and it has not proven so efficient as excision of the superior cervical ganglion for the reduction of intra-ocular tension. It should be considered not as an alternative of iridectomy or even of sympathectomy, but rather in comparison with opticociliary neurotomy or excision of the eyeball.

Pathology.—While the effects of increased or diminished functional activity in the sympathetic nerve are well understood, the recorded observations as to its pathological histology are so inconclusive as to furnish no basis for a classification of its diseases. Indeed, they here require but little notice. Weeks[4] finds regarding glaucoma: "The testimony in our possession is not sufficiently conclusive to enable us to say that there is any constant change in the cervical sympathetic peculiar to glaucoma. Nor is it sufficiently conclusive to exclude the possibility of such constant change." The most constant change found in the ganglia removed has been an increase of pigment in the neuron cell-bodies. But this may be a senile change. Increased connective tissue, or sclerosis, which has been reported by several observers, has not been found at all by others equally competent.

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[1] Trans. Sec. on Ophthalmology, A. M. A., 1903.

[2] Ibid.

[3] Annales d'Oculistique, July, 1902.

THE SECRETION AND REMOVAL OF TEARS.

Secretion.—The control of nerve impulses, either of reflex or of central psychic origin, over the secretion of tears is one of the commonest observations in ocular physiology. It is also certain that reflex lachrymation is especially likely to arise from impressions made through the fifth nerve, as by the irritation of wind or dust in the eye, pungent gases, etc. But the exact channels by which the efferent nerve impulses governing its action reach the lachrymal gland have not been determined.

While fibres of the sympathetic nerve enter the gland along each of its nutrient vessels, its most evident nerve-supply is through branches of the fifth nerve. Chiefly it is supplied through the lachrymal branch of the ophthalmic division, which runs forward near the roof of the orbit to be distributed to the gland. But anastomosing with this is a twig coming from the orbital branch of the second division of the trigeminus; and this anastomotic twig may have special importance with reference to the lachrymal secretion. A large part of the lachrymal branch of the first division passes through the gland to be distributed to the conjunctiva and skin adjoining. (See Fig. 8.)

The results of physiologic experiments are so contradictory, possibly on account of the variations of function of the different animals experimented upon, that they throw no certain light upon the neurology of the lachrymal secretion. They need not be here discussed, except to point out that increased secretion of tears after section of the sympathetic or after removal of the superior cervical ganglion is usually quite temporary, and probably depends upon vasomotor paralysis.

When it comes to actual disease of the nerves, lesions of the fifth nerve seem less frequently to cause disturbance of lachrymation than those of the seventh. In trigeminal neuralgia there is no constant or characteristic alteration of the lachrymal secretion. Nor is any lachrymal disturbance more certain to occur, with neuralgia involving the first division of the nerve, than when the second or third division is affected, without the first. The intense pain may cause the increased lachrymation, very much as pain elsewhere, or irritation of the eyes or nasal mucous membrane may cause it. A more specific disturbance of the lachrymal function has, however, been noticed by Uhthoff in one case of peripheral neuritis affecting the first and second divisions of the fifth nerve.

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[4] Trans. Sec. on Ophthalmology, A. M. A., 1903.

Paralysis of the seventh nerve, by relaxation of the orbicularis, allowing the lid to fall away from the eye-ball, commonly disturbs the removal of tears. But in a considerable number of reported cases, facial paralysis has clearly been attended with diminution, if not complete cessation, of the secretion of tears on the affected side. Probably the symptom would be more frequently noticed if the diminished removal of tears did not mask the diminished secretion. The interference with secretion, and the interference with removal, to some extent neutralize each other; and render the lachrymal disturbances attending the seventh nerve lesions less noticeable than they otherwise would be.

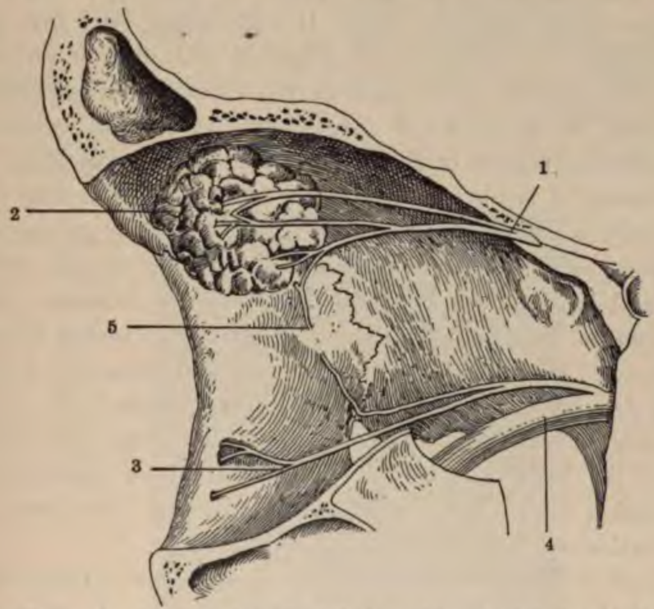

FIG. 8.—Nerve supply of lachrymal gland. 1, lachrymal branch; 2, lachrymal gland; 3, malar branch; 4, second division of fifth nerve; 5, anastomotic branch connecting gland with second division. (Merkel.)

While the neurology of lachrymation cannot be regarded as settled, the share of the seventh nerve in the control of the gland might be explained in this way: As has already been pointed out (page 333), the seventh nerve gives off at the geniculate ganglion the petrosal branches; the superior, or greater, petrosal passes to the sphenopalatine ganglion, which gives branches to the second division of the trigeminus; and this, as we have seen, supplies an anastomotic branch to the lachrymal nerve. Through this channel efferent impulses, emerging from the nerve-centres by way of the seventh nerve, may reach the lachrymal gland. The similarity of the arrangement with that of the chorda tympani, given off from the seventh more peripherally than the great petrosal, and joining the third division of the trigeminus, is quite striking. That the chorda tympani exerts an important influence over the secretion of the salivary glands is well known, and this view of the nerve-control of the lachrymal secretion adds one to the striking analogies that may be traced between the functions of salivation and lachrymation. It should not be forgotten, however, that definite proof regarding the innervation of the lachrymal gland is yet lacking.

The amount of lachrymal secretion formed in a given time depends almost wholly upon reflex influences. Thus, Schirmer points out that the secretion practically ceases during sleep. Under psychic influences or peripheral irritation, it is capable of enormous increase. Even in those persons in whom epiphora is most constant it occurs in response to some especial stimulation, as from chronic inflammation of the lachrymal sac, eye-strain, or the ordinary exposures, which in the particular patient cause an excessive reflex. It is a significant fact that after removal of the lachrymal sac for dacryocystitis, the patients usually have no further trouble from epiphora, showing that it has been a reflex of the irritation of the diseased tear passages.

Increased or diminished lachrymation may occur in organic disease of the central nervous system, as locomotor ataxia. In exophthalmic goitre increased lachrymation has usually been ascribed to exposure of the conjunctiva by retraction of the lids and defective winking. But Berger has pointed out that it may antedate the symptoms of Dalrymple and Stellwag (See Pages 668 and 840).

Increased or diminished lachrymation may occur in organic disease Schirmer, from his very careful study of the subject, concludes that in the act of winking, tension upon the palpebral tendon causes a dilatation of the lachrymal sac, and the sucking into it of tears from the conjunctiva. When the eye is reopened the elasticity of the sac forces the tears on into the nose. Probably an important part in the process is taken by the minute sphincter muscles which surround each lachrymal punctum. Relaxation of these sphincters during winking favors the passage of the tears into the canaliculi; and their contraction at other times tends to prevent regurgitation.

The abnormal, continuous relaxation of this little sphincter muscle, either by chronic disease or forcible dilatation, impairs the natural ability to remove any excess of lachrymal secretion. In order that the tears shall enter the canaliculi the puncta must be turned in towards the eyeball, and the lids be in contact with the globe of the eye, so that the tears shall be conducted by capillary attraction to the puncta. Paralysis of the orbicularis causes interference with the passage of the tears by the inability to maintain these latter conditions.

THE LIDS AND THEIR MOVEMENTS.

The position and form of the lids and palpebral opening respond so clearly to nerve impulses and to such varied impulses that they afford diagnostic evidence of great value. But they are subject to marked normal and pathologic variations, due to alterations in the tissues of the lids themselves or of the structures that they cover, and these variations must be thoroughly understood before the symptomatic value of the appearances presented can be estimated.

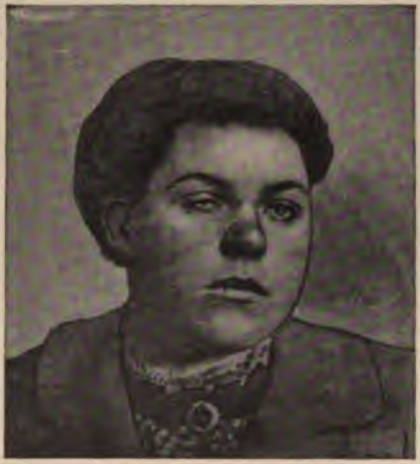

FIG. 9.—Thickening of the lids produced by trachoma. (Case of Dr. Jackson.)

The lids form a freely movable curtain stretched across the orbital opening, loosely attached in all directions except to the nasal side in the region of the internal palpebral ligament. They are usually comparatively thin and flaccid except for the normal elasticity of the skin and other tissues composing them. They lie upon the deeper tissues from which they largely take their outline. But the lids may become thickened so that the outline of the deeper structures is quite masked. Normally comparatively free from fat in the adult, they may become the seat of extensive deposits of adipose tissue, and usually do contain a good deal of it in early childhood. Their loose cellular tissue favors their rapid and excessive infiltration with serum, and they may become greatly thickened with more substantial and permanent exudate.

Still more confusing are the changes produced by inflammation. These are quite common as a result of trachoma. In the chronic cases the deposits of connective tissue, besides thickening, bind the lid rigidly to adjoining tissues, greatly interfering with all of its movements. The effect produced is illustrated in Fig. 9.

In this patient the alterations produced by chronic trachoma were chiefly confined to the right eye, so that the left serves as a standard of comparison. Syphilitic tarsitis causes a somewhat, but not exactly, similar alteration of appearance and function. It is generally bilateral.

In the curtain of the lids the features especially influenced by nerve impulses are the palpebral opening and the superior and inferior orbitopalpebral folds. Of these folds the lower, between the lower lid and margin of the orbit, is most marked when the eye is turned strongly down. But even with that movement it may be quite indistinguishable, especially in children with much fat in the lids. On looking up there remain only the lines or creases, usually two, which mark the site of the fold. Its formation evidently depends upon traction of the inferior rectus, through its connections with the lid (Fig. 11). These folds are shown in Fig. 10.

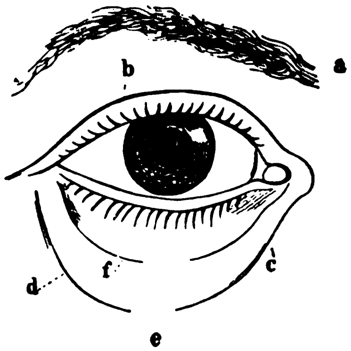

FIG. 10.—Palpebral folds. *a*, brow: *b*, oculo-orbital fold, superior; *c*, *d*, *f*, inferior folds; *e*, cheek. (Wilbrand and Saenger.)

The upper fold *b* is well marked, although it varies greatly in appearance and becomes much deeper when the eye is rolled upward.

When the orbit is well filled with fat, it appears merely as a close fold in the skin. But when the adipose tissue is removed by age or wasting disease it becomes a broad fossa, which is the chief factor in producing the appearance of hollow or sunken eyes, in wasting disease. Below there are generally several smaller creases, *c*, *d*, *f*, divided into a temporal and a nasal group, which become emphasized as the eye is turned down and from or toward the nose.

For a clear understanding of alterations brought about in the lids and palpebral opening, one must bear in mind the anatomic and physiologic relations of the various structures of the orbit. To the margin of the orbit is attached a sort of connective tissue hammock, including part of the capsule of Tenon, denser connective tissue bands, and looser meshes. In this hammock lies the eyeball, drawn firmly back by the elastic tension of the recti muscles, and rotated or swung by their individual or combined contractions. The globe of the eye is not a ball turning in a fixed socket. The well-known fact that the centre

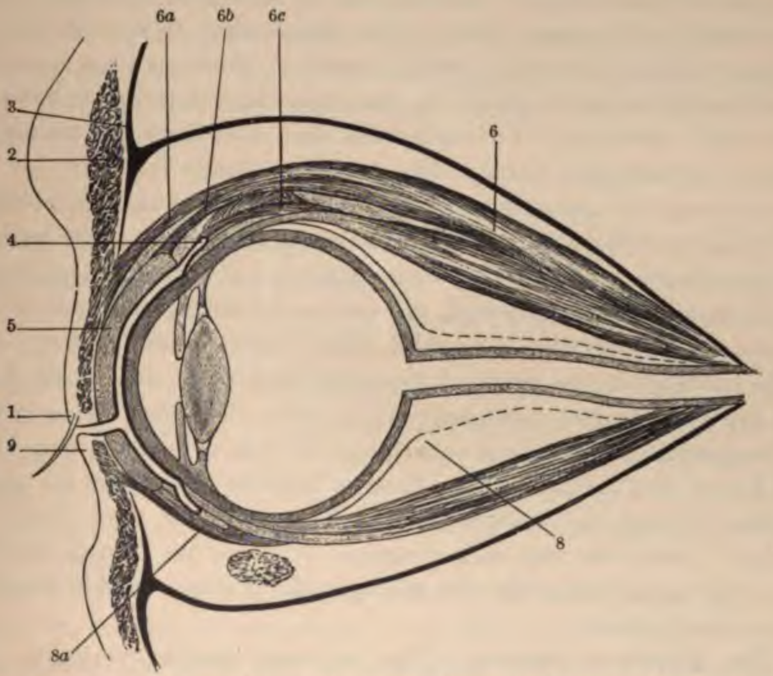

FIG. 11.—Section of orbit. 1, skin of lid; 2, orbicularis muscle; 3, tarso-orbital fascia; 4, conjunctival cul de sac; 5, tarsus; 6, elevator of upper lid; 6*a*, unstraited muscle connecting elevator with skin of lid; 6*b* and 6*c*, connections of elevator with tarsus and conjunctiva; 7, skin of lower lid; 8, inferior rectus; 8*a*, connection of lower lid. (Schwalbe).

of rotation is back of the centre of the eyeball means that the eyeball as a whole moves somewhat in the direction toward which it is rotated. On looking up it rises; on looking down it is depressed; on looking to the right or left it moves perceptibly in these directions. All such movements of the eyeball are closely followed by movements of the lids. The parts concerned are illustrated in Fig. 11.

The harmony of movement of eyeball and lids is aided by the numerous bands that pass from the tendons of the recti and inferior oblique muscles to the lids; so that the lid movements are partly due to the action of the muscles which turn the eyeball.

A good example of how such harmony of movement is brought about is revealed by a careful study of the connections of the elevator of the upper lid and the superior rectus muscles. Besides a close anatomic association of the nuclei governing these two muscles and their habitual physiologic association of simultaneous use, slips from the tendon of insertion of each pass to the tendon of insertion of the other.

Harmony of movement even of different parts of the upper lid is secured by such an arrangement of muscular insertions. The part of the skin covering the cartilage of the upper lid is normally kept rather closely applied to it, although the skin and cartilage are not firmly connected except at the lid margin. This close apposition of the skin to the cartilage is preserved, whatever the position of the lid and however great the general redundancy of the skin. This has been ascribed to the elasticity of the tissue connecting the skin with the tarsus. But it is known that certain anterior fibres of Mueller's muscle in the upper lid pass through the orbicularis to be inserted in the skin of the lid. Dwight[1] points out that their function is to draw the skin to the fold above the tarsus when the lids are opened. Failure of this function causes atonic ptosis.

The Palpebral Opening.—The palpebral opening varies in size and shape from a mere slit separating the closed lids to an almost circular aperture, the diameter of which approaches that of the eyeball. The active factors in producing these changes are the constriction of the orbicularis tending to diminish the opening, and the contractions of the elevator of the upper lid and the superior and inferior recti muscles, with the attached unstriated muscles, which tend to retract the lids and so enlarge the opening. Passively, narrowing of the opening may be due to inflammatory or other deposits which tend to oppose the retractors of the lids or to weakness or paralysis of these retractors.

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[1] System of Ophthalmology, edited by Norris and Oliver, vol. i. p. 92.

Widening of the palpebral aperture is passively favored by paralysis of the orbicularis, or by enlargement of the eyeball, or its being pushed forward by pressure from behind, or by cicatricial adhesions preventing the normal closure of the lids.

The nerves directly concerned in the changes of form of the opening are the facial, distributed to the orbicularis; the oculomotor, supplying the recti and the elevator of the upper lid; and the sympathetic, which is supposed to innervate the unstriated fibres of Mueller's muscles of the lids. The palpebral opening is generally more nearly circular and relatively wider in childhood. Excitement and probably the influence of general cold tend to widen the aperture. Stimulation or irritation of the cervical sympathetic and the local application of cocaine have the same influence. On the other hand, age, weariness, stupor, paralysis of the sympathetic or its division or excision, tend to diminish the palpebral opening.

Exophthalmos widens and enophthalmos causes narrowing of the aperture. But retraction of the lids and widening of the palpebral opening causes an appearance of exophthalmos, while narrowing of the opening causes an appearance of enophthalmos. Unfortunately, the prominence of the eye-ball in the orbit and the actual variations in its position with reference to the plane of the orbital opening have rarely been subjected to actual measurement; so that there may often have been retraction of the lids causing an appearance of exophthalmos, when it was supposed that widening of the palpebral opening had been caused by exophthalmos. Certainly, eyes that appear to be protruded or retracted often prove not to have changed their relations with the orbital margin.

On the other hand, actual protrusion of the eyeball may be accompanied by swelling or relaxation of the lids leading to narrowing of the lid opening, which partly masks the exophthalmos. In cases of exophthalmos slowly developed by the increase of an orbital tumor the lids may be enormously enlarged.

Winking.—Normal winking is a rhythmical closure of the palpebral opening by the contraction of the tarsal portion of the orbicularis, as a reflex probably to a need for renewed moisture of the corneal surface, a sort of thirst of the part, which usually does not affect consciousness. The afferent impulse passes through the fifth, the efferent through the seventh nerve. Winking is repeated from two to five times a minute. Howe,[1] who studied it by photography, finds that the closing of the lids occupies one-twentieth to one-tenth of a second. They remain closed two- or three-tenths of a second, and the lifting of the lid again takes one- or two-tenths of a second. Slow lifting, he thinks, may be a valuable sign of paresis of the elevator of the lid.

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[1] Transactions of the American Ophthal. Society, 1904.

Lid Signs.—Although the normal variations in the palpebral opening make it difficult to judge at first sight whether or not the lids are abnormally separated, any change of the kind is likely to be noticed, in the expression of the eyes, by those familiar with the patient. When the lids are separated too widely it gives the eye a staring expression. When this is habitually present the retraction is probably due to spasm of the unstriated muscle-fibres of the lids innervated through the sympathetic. This has been called *Dalrymple's sign*. It may cause exposure of the sclera both above and below the cornea. It can be imitated by the application to the eye of cocaine, which produces a similar spasm of Mueller's muscle that lasts considerably longer than the anæsthesia, but hardly so long as the dilatation of the pupil produced by this drug. (Compare Page 835.)

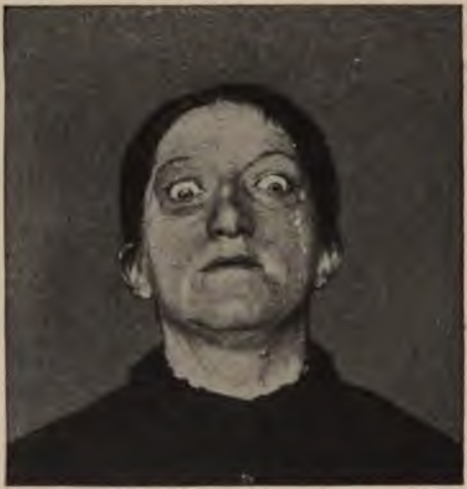

FIG. 12.—Graefe's sign: exposure of sclera on looking down. From a case of exophthalmic goitre. (Wilbrand and Saenger.)

The normal movements of the lids with the eyeball have already been referred to. The movements of the upper lid, upward or downward as the eye is turned up or down, is by far the most extensive of these lid movements, and in health is very accurately co-ordinated with the movements of the globe. In disease the downward movement of the lid, when the eye is turned down, may become very defective or altogether wanting. The upper lid remaining elevated and retracted when the eye is turned downward causes an appearance that is strikingly abnormal. This is shown in Fig. 12.

This is named for its discoverer, *the Graefe sign.* It is easily confused with the Dalrymple sign. When the eyes are turned down they are really too widely open. But if the Graefe sign alone be present it can be seen that there is no special retraction of the lower lid; and when the eyes are turned up there is no abnormal widening of the palpebral opening. In practice the two signs are very likely to be seen together, there being both a widening of the opening—Dalrymple's sign—and a failure of the upper lid to move down with the eyeball—Graefe's sign. The disturbance of the co-ordination of the lid movement and eye movement is probably a failure of function in the nuclei controlling these movements; or the effect of some abnormal impulse or lack of control from the higher centres. (Compare Page 833.)

An impairment of a closely related function, a disturbance of the nervous mechanism concerned in the reflex movements of the lids, leads to a third lid sign named for Stellwag. *Stellwag's sign* consists of infrequent, irregular, and incomplete winking, occurring independently of any paresis of the seventh nerve or defective sensibility in the cornea or conjunctiva. The eye exhibiting it may remain a minute or more without winking. Then there may be repeated inperfect winking movements. Sometimes the movements will be repeated at nearly the usual intervals (two to five times a minute). But most of the closures of the eye will be incomplete. Where Stellwag's sign is well marked the lid movement of winking will be noticeably hesitating and irregular in character. The sign of Stellwag is commonly associated with those of Graefe and Dalrymple. (Compare Page 834.)

Lid Reflexes.—The reflex closure of the lids to a touch of a foreign body upon the cornea or conjunctiva felt through the fifth nerve, or to the impression of something approaching the eye transmitted through the retina and optic nerve, is very prompt, certain, and quite beyond the control of the will. Any one who frequently has to instil fluids into the eye or make applications to the conjunctiva will appreciate the constancy and force of this reflex. Touching or striking the skin of the lids or neighboring parts of the face will produce a movement toward closure of the palpebral opening.

A special sensorimotor reflex of the lids has been described by McCarthy, who calls it *the supraorbital reflex.* To elicit this reflex the patient should relax the muscles about the eyes. Then a slight tap of the percussion hammer is given high on the forehead. This usually excites a general movement of closure of the lids. But the action to which McCarthy calls attention is a quick fibrillary contraction of the lower half of the orbicularis palpebrarum. This reflex is absent after division, or disease destroying the function of the orbital nerve, and in all cases of peripheral paralysis of the facial nerve. (See Page 916.)

Widening of the Palpebral Opening by Contracture of the Elevator.—Besides the opening of the eye produced by the healthy exercise of the function of the elevator of the upper lid and that due to retraction of the lids by Mueller's muscle, which is discussed elsewhere, abnormal widening of the palpebral fissure may occur as a reflex or choreiform symptom. Posey [1] has reported a case of rhythmical widening about fifteen to twenty times a minute, which ceased after the use of atropine for the measurement of ametropia. Other cases of similar peculiar tremor of the lids have been reported in connection with exophthalmic goitre, ophthalmoplegia externa, and facial paralysis. Secondary contracture of the elevator of the upper lid sometimes occurs after facial paralysis of either peripheral or central origin. It leads to abnormal widening of the opening, closely resembling the Dalrymple sign.

Lagophthalmos.—Continuous exposure of the eyeball by inability to close the lids may be due to cicatricial contraction deforming the lids or drawing them away from their normal position and binding them down so as to interfere with their function. In such cases the scars responsible for the deformity are immediately evident upon inspection.

In elderly people with great relaxation of tissue and general muscular weakness, there may be inability to close the eyes fully. In cases of this kind there is often some conjunctival inflammation and swelling, so that ectropion of the lower lid occurs and may give more trouble than the exposure of the eyeball.

Complete facial paralysis of peripheral origin is always attended with inability to close the eye by voluntary contraction of the orbicularis, although the lids can easily be drawn together with the fingers. With attempts at forcible closure of the lids, however, the eyeball is normally rolled up so that the cornea is greatly protected from injury; and in many cases the inability to close the lids by winking is replaced by frequent movements of the eyeball upward, which secures the moistening of the cornea and removal of foreign bodies from its surface. If, however, these movements are not made with sufficient frequency, or, if in a case of long standing, secondary contracture of the upper lid increases the exposure of the eye-ball, there is great danger of serious disease of the cornea. This is particularly likely to occur in elderly or poorly nourished patients or when the sensibility of the cornea is impaired.

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[1] Journal of Nervous and Mental Disease, July, 1902.

Exposure Keratitis.—Lagophthalmic keratitis arises from the undue drying of the exposed portion of the cornea. It begins with disturbance of the epithelium and superficial ulceration near the lower corneal margin. Infection occurring through the channels thus opened to it leads to purulent infiltration of the subjacent tissue. The infiltration runs a comparatively chronic course and is marked by slight inflammatory reaction, as compared with that which usually attends an infective keratitis of equal extent and gravity. If unchecked, the process goes on to perforation of the cornea, extensive adherent leucoma, and practical loss of usefulness of the eye.

The treatment of this condition must include careful protection of the eye from irritants, as by dressings that will close the lids and hold them shut. If corneal infection requiring active treatment has not occurred or has been overcome, the most efficient means of protecting the eye is by uniting the margins of the lids. These may be freshened and brought together by sutures throughout a part of the palpebral fissure. Usually the tarsorraphy is done at the outer canthus, but in some cases a better effect will be obtained by uniting the lid margins near the region of the lachrymal puncta. In this way we secure the proper drainage of tears and leave the temporal portion of the palpebral opening free, preserving part of the field of vision. The exact extent and location of the union of the lids must be determined by study of the individual case. If union of the lids be resorted to early, before infection has occurred, the prognosis is quite favorable. And if at a subsequent time recovery of the power of the orbicularis occurs, the connection between the lids can readily be divided.

Ptosis.—The conditions apart from abnormal innervation which cause narrowing of the palpebral opening have already been referred to (page 347). Congenital ptosis, hysterical ptosis, and ptosis due to disease of the oculomotor nerve, or of the central nervous system, are discussed in Chapter V. It remains here to consider the general characteristics of ptosis, a few forms arising from local conditions, and a brief outline of the treatment. In a general way ptosis is due to relaxation of the elevator of the lid or to a more or less permanent contracture of the orbicularis.

Relaxation of the elevator is attended with comparative smoothness of the skin of the lid and partial obliteration of the upper oculo-orbital fold (Fig. 13). Complete paralysis of the elevator is shown by the fact that on looking upward there is no deeping of this fold. Contraction of the orbicularis tends to cause wrinkling of the skin of the lids and adjoining parts.

Temporary Ptosis.—Any inflammatory disease of the eye which continues many days is liable to cause narrowing of the palpebral opening. This occurs quite independently of any exudation into the lids that might mechanically oppose their full opening. It is at first an effort to protect the inflamed eye from light or irritation. It is most noticeable in connection with chronic inflammation affecting but one eye. Such a ptosis may be continued indefinitely to diminish the light falling on a nebula left by corneal inflammation. But even when no such sequel remains and the inflammation has entirely passed away, nerve habit may continue for a time a drooping of the lid. This drooping, however, will slowly diminish, and in the course of weeks or months will entirely disappear. The process of recovery may be hastened by massage and voluntary efforts to open the eye widely. Possibly electric stimulation of the muscle may be of benefit. But such ptosis may be due to contracture of the orbicularis rather than to relaxation of the elevator, and injudicious use of electricity in such a case would be pretty certain to do more harm than good.

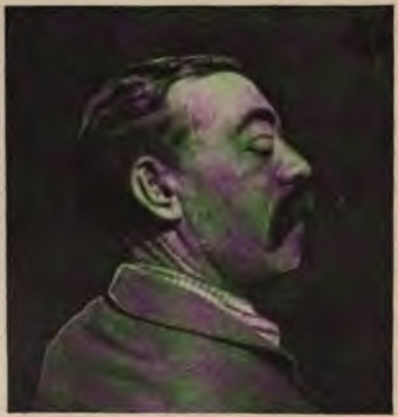


FIG. 13.—Ptosis: obliteration of palpebro-orbital fold. (Case of Dr. Jackson.)

Another form of temporary ptosis is seen in persons with organic disease of the central nervous system. Sometimes the upper lid will droop for a few hours, sometimes for days or weeks, although the patient can usually open one eye widely by special effort and there may be no other evidence of oculomotor paresis. This form depends on relaxation of the elevator. The prognosis regarding it is very uncertain, but it may disappear permanently, even after it has been troublesome for a long time.

Not a few persons complain of special difficulty in opening the eyes after particularly sound sleep. Although there is no adhesion of the lids and the patient seems to have fully regained consciousness, there remains a sense of inability to move the lids. This may be compared with the feeling experienced in a nightmare, of inability to perform some act in spite of the strongest desire to do it. The specially intimate connection of closure of the eyes with the phenomena of sleep is probably the basis of this kind of ptosis.

Atonic Ptosis.—Without loss of power in the elevator of the upper lid, and with normal movement of the tarsal cartilage, the skin of the lid may form a fold hanging down over the lid margin and roots of the lashes, so as to limit materially the field of vision. This condition may be simulated by a redundancy of tissue. In the older works it was called ptosis from hypertrophy, while atonic ptosis (Mackenzie) was used to designate what would now be known as hysterical ptosis. Anatomical examination of the most typical cases of this class has shown atrophy rather than hypertrophy of the tissue. Hotz pointed out that the condition was due to impairment or destruction of the normal relation between the skin of the upper lid and the tarsus. So that when the tarsus was retracted the relaxed skin formed a fold which fell over its margin. The connection which Dwight has pointed out, between a part of the skin and a slip of Mueller's muscle connecting it with the tendon of the elevator, suggests that this form of ptosis may be due to relaxation of this muscle, and properly rank as a form

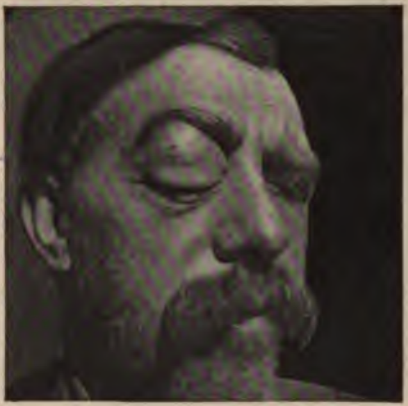


FIG. 14.—Swelling of lids following traumatism. (Case of Dr. Jackson.)

of ptosis from disease of the sympathetic. Its treatment will be mentioned under the operative treatment of ptosis.

Traumatic Ptosis.—Scars involving the lids may cause ptosis, but this form need not be described here. Attention is called to it that inability to raise the lids from this cause shall not be confused with that due to impaired nerve action. Penetrating wounds of the orbit may involve the branch of the oculomotor nerve which supplies the elevator muscle. But they are more likely to do direct injury to the muscle. If the wound be extensive the involvement of the muscle in the resulting scar is likely to produce permanent disability. Ptosis from this cause is illustrated in Fig. 14. Some power of movement may be regained with the subsidence of the inflammatory swelling, but some permanent impairment is the rule after such injuries.

Operative Treatment of Ptosis.—Without discussing the numerous ptosis operations in detail, they may be mentioned with the principle involved in each. For traumatic ptosis a plastic operation, planned to meet the indications of the particular injury, may be required. The object aimed at, in such an operation, will be the freeing of the lid from abnormal restriction of its upward movement.

For ptosis due to hypertrophy either of the skin of the lid or subcutaneous tissue, the removal of the proper quantity of hypertrophied tissue is the rational procedure. But for ptosis not due to actual hypertrophy, even for that which is evidently associated with great relaxation, the mere removal of tissue is likely in the end to prove unsatisfactory. Excision of an elliptical piece of skin may be of some benefit in the slighter degrees of paretic ptosis. But contraction thus brought about tends to obliterate the fold between the lid and upper margin of the orbit, causing a deformity as noticeable as that which it removes. Excision of an excessively large flap, in addition to causing this new deformity, may produce a dangerous lagophthalmos, while it still fails to remove the ptosis on turning the eye upward.

A more reliable operation and one that does not cause the same obliteration of the oculomotor fold is that of Wilder. He exposes the tarso-orbital fascia by making an incision just below the brow and dissecting a flap down to the tarsus. Sutures introduced through the margin of the tarsus are carried in and out through the fascia, as gathering stitches, up to the line of incision, drawn upon sufficiently to give the proper elevation of the lid, tied and cut short. The incision is closed by skin sutures. This gathers or folds the fascia upon itself, and leaves an almost imperceptible scar.

Atonic ptosis, which has often been taken for hypertrophic and subjected to excision, should be treated as suggested by Hotz. An incision in the general direction of the lid margin, two or three millimetres from the margins at its ends, and somewhat farther from the margin in the centre, is made through the skin and down to the cartilage. This incision is closed by sutures which pass first through the lower lip of the incision and then through the upper margin of the tarsus. These sutures when tightened stretch the lower flap of skin and unite it firmly to the tarsus, so that the subsequent folding or falling below the lid margin would be impossible.

For ptosis due to paresis of the elevator, operations for the advancement, resection, or folding (tucking) of the tendon of the elevator are appropriate. In Eversbusch's folding operation the tendon is exposed four millimetres above its insertion into the tarsus. A suture with a needle on each end is applied to the tendon, and the needles carried between the skin and tarsus to the lid margin, where they are brought out and the ends of the suture tied over a bit of rubber tubing. Three of these sutures are placed: one at the centre and one at each end of the lid. The tightening of these sutures folds the tendon down over the tarsus.

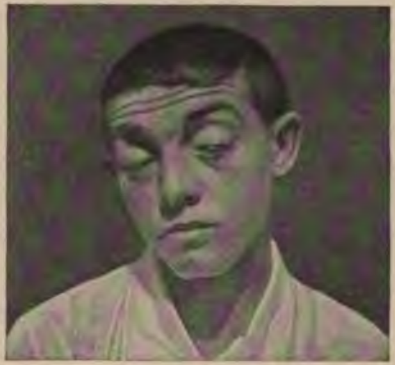


FIG. 15.—Complete bilateral ptosis. Maximum elevation of the lids by contraction of the frontalis. (Case of Dr. Jackson.)

Snellen's operation is a resection. The tendon is exposed and three or four needles thrust through it the necessary distance back from the tarsal margin. The end of the tendon is then excised by parallel incisions along the tarsal margin and in front of the needles. The needles being brought through the upper margin of the tarsus, and the threads tightened, the desired shortening of the tendon is secured.

Wolff does resection by exposing the tendon and raising it on two strabismus hooks. The sutures are introduced through the tendon just below the upper hook. The end of the tendon is then cut away from the tarsus, and the sutures passed through the insertion of the tendon into the tarsus.

De Lapersonne practises advancement of the tendon of the levator, with or without excision, by suturing the tendon upon the outer surface of the tarsus instead of to its upper margin.

For paralysis of the elevator which is complete and permanent, the only operations worthy of much attention are those which give power to raise the lid by attaching it to some other muscle which may thus be made to perform a vicarious function. The effort of nature in these cases is to drag the lid upward through its skin connection with the frontalis. The result achieved is shown in Fig. 15.

To give a more firm and efficient connection between the lid and frontalis, Dransart sought to produce a scar by introducing cat-gut sutures subcutaneously from the lid to the brow. To increase the cicatricial change, Pagenstecher substituted silk for the catgut. This was tied over a bit of rubber tubing above the brow, allowing it to be shortened from time to time as seemed desirable. Mules, finding the scars secured by such measures left much to be desired in the way of permanence and rigidity, introduced a silver wire by which the desired elevation of the lid was secured. The wire was then knotted and the knot buried above the brow. Worth has used kangaroo tendon for this purpose. Harman, instead of silver wire, has employed the light, woven gold chain sometimes used for an eyeglass-cord. This being thoroughly sterilized and buried becomes intimately incorporated with scar-tissue, and, being entirely flexible, is less likely to cause subsequent disturbance than is the silver wire.

The connection of the lid and frontalis tendon by transplanted tissue was effected by Panas in the operation which bears his name. In this operation an incision is made parallel to the brow above it and another below. The tissue between them is undermined. A broad, tongue-shaped flap of skin and subcutaneous tissue is dissected up from the lid and oculomotor fold, carried up under the bridge of tissue, and made fast at the insertion of the frontalis muscle into the skin of the brow.

Panas's operation has been the subject of slight modifications by individual operators; but is essentially the operation most commonly done to-day for this form of ptosis. All operations that aim to connect the lid with the frontalis tendon, whether by thread, wire, scar-tissue, or flap, have these defects: the oculo-orbital fold is more or less obliterated and the movement of the lid is at best accomplished by an abnormal contraction of the frontalis.

The muscle which normally contracts in closest association with the elevator of the upper lid is the superior rectus. In a normal state every contraction or relaxation of the one muscle is accompanied by a similar contraction or relaxation of the other; except that when the eye is strongly closed the elevator of the lid is relaxed, but the eyeball is rolled up by contraction of the rectus.

Motais devised the operation of connecting a portion of the tendon of insertion of the rectus with the tarsus. An incision being made through the conjunctiva and subconjunctival tissue down to the tendon of the superior rectus on the eyeball, and in the direction of the tendon, is carried back to the retrotarsal fold and through the palpebral conjunctiva to the tendon of the elevator of the lid. The rectus tendon is isolated, split into three parts, and the middle part separated from the insertion into the sclera and by two sutures carried up and inserted in the upper margin and external surface of the tarsus.

Thus treated, the superior rectus muscle quickly gains power to move both the lid and eyeball. So that henceforth the lid moves normally with the globe of the eye, except that on forcible closure of the lids the upper lid is pulled up with the effort of the superior rectus to roll the eye upward. Motais's operation causes no visible scar and tends to restore to normal the oculo-orbital fold. It gives to the lid its most important and useful movements. But it does not confer the power of retraction of the lid independently of movements of the eyeball.

CHAPTER VIII.

DISEASES OF THE RETINA AND OPTIC NERVE.

BY H. V. WÜRDEMANN, M. D.

OPHTHALMOSCOPY.

SINCE the possibility of examining the interior of the eye by means of the ophthalmoscope was demonstrated by von Helmholtz in 1851, much light has been thrown, by the use of this instrument, upon various general as well as ocular diseases, and the diagnosis of many general disorders and diseases of the nervous system has been greatly simplified. Though the instrument, as originally devised by von Helmholtz, possessed all the properties essential to a thorough examination of the fundus, its elaboration and improvement by Reute, Liebreich, Loring, and Morton, have added greatly to the facility with which it may be

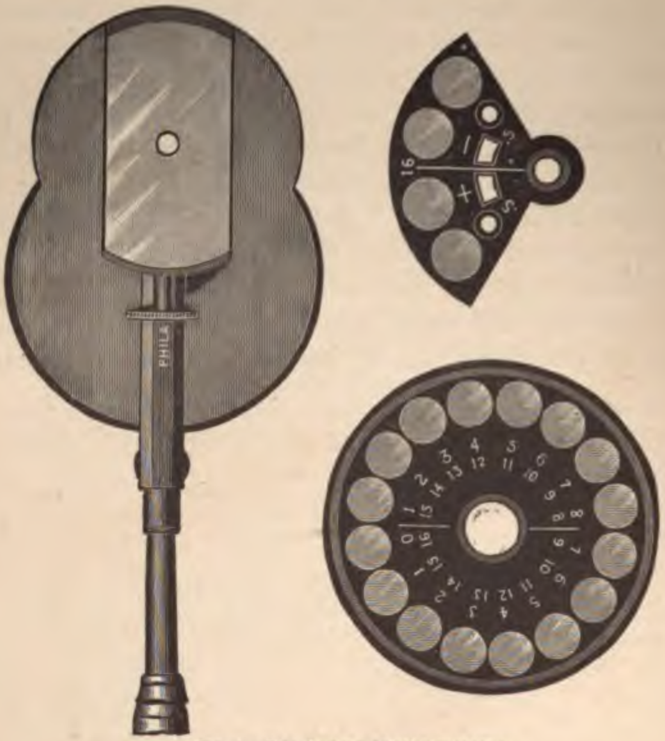


FIG. 1.—The Loring ophthalmoscope.

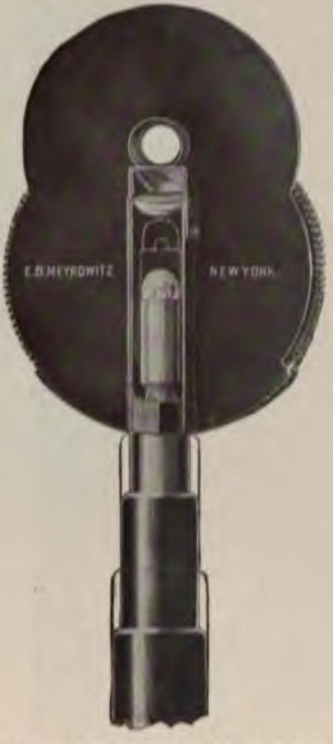


Fig. 2.—The Meyrowitz electric-lighted ophthalmoscope.

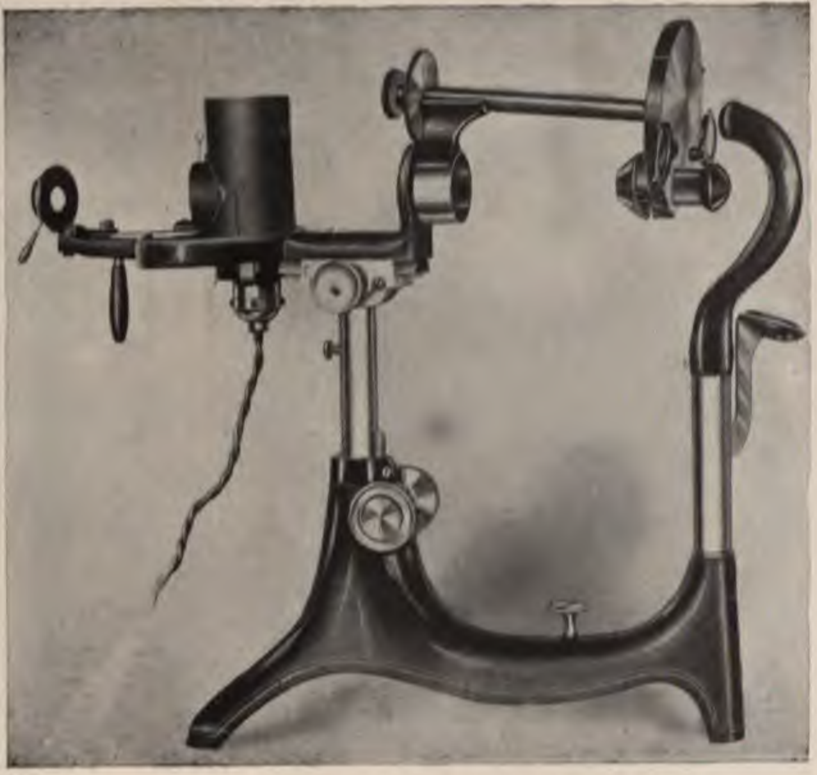


Fig. 3.—Combined stationary ophthalmoscope and retinoscope. (Geneva.)

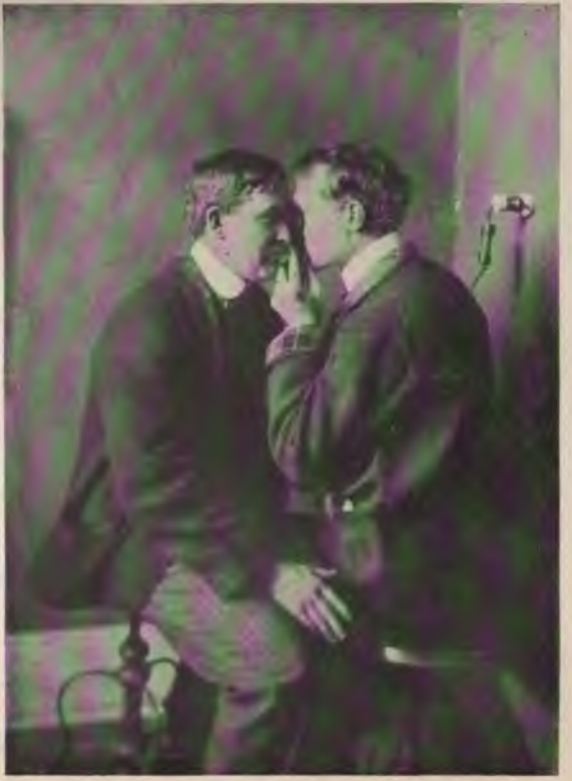


FIG. 4.—Ophthalmoscopy. The direct method with electric ophthalmoscope.

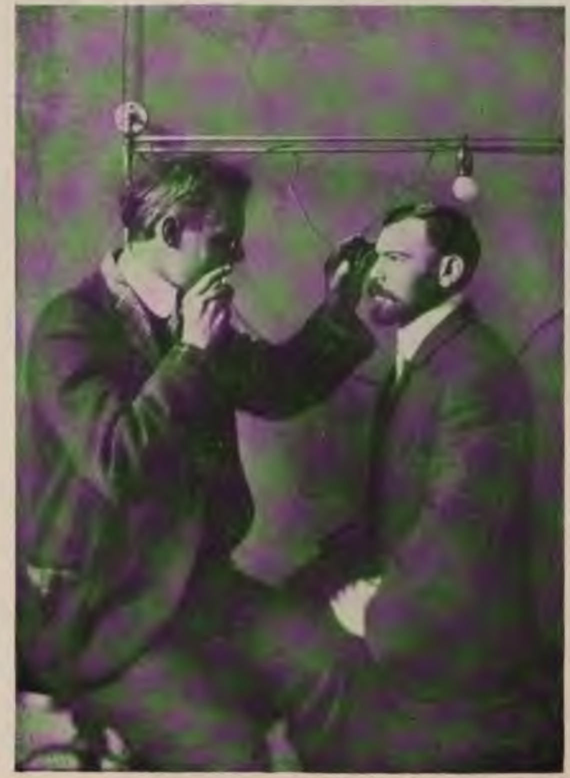


FIG. 5.—Ophthalmoscopy. The indirect method with reflecting ophthalmoscope.

used and have rendered it possible for the expert to estimate very accurately the refraction of the eye.

The accompanying cuts illustrate the Loring ophthalmoscope (Fig. 1), the most popular instrument in this country, and a model of an electric-lighted ophthalmoscope (Fig. 2), which is especially serviceable for bedside work. Fig. 3 illustrates a model of a stationary ophthalmoscope. This instrument is a valuable adjunct to the hand ophthalmoscope, as it gives an extended view of the magnified and inverted image, is most useful for demonstration to students, and for the purpose of reproducing fundus pictures by drawing or photography.

In ophthalmoscopy the illumination should be steady and uniform, and while the fundus may be seen on bright days with ordinary daylight passing through a small aperture into a darkened room, this source of illumination can be used only occasionally, and thus most of our conceptions of the fundus are based upon observation by yellow artificial light; a candle or lamp flame, a gas jet or an electric light covered by frosted glass, being utilized for the purpose. In the performance of all ordinary tests with the ophthalmoscope, both the observer and the patient should be comfortably seated. If the patient be bedridden this is impossible and the examination must often be conducted from a constrained posture. Although this is often fatiguing, the surgeon should not desist until he has satisfied himself that he has accomplished all that was possible under the circumstances.

The interior of the eye may be viewed with the ophthalmoscope by either the *direct* or *indirect* method. The former is to be preferred, for although somewhat more difficult of performance, it permits of a study of the fundus under a much greater magnification, the back part of the eye being magnified 14 diameters by the direct method, and but 4 by the indirect, and at the same time it enables the observer to obtain a very accurate idea of the refraction of the eye. The indirect method is only to be preferred in high myopia and in cases where the media of the eye are hazy. Before resorting to the study of the interior of the eye, the observer should always obtain as much information as is possible of the media by means of *transmitted light.* For this purpose, the observer, at a distance of about 30 centimetres, flashes the light which is placed behind and slightly to the side of the patient's head into the eye, by means of the mirror of the ophthalmoscope. A faint pinkish-red glow will be seen to replace the blackness of the pupil. This is known as the fundus reflex, and if the glare be unbroken and uniformly bright, even when the eye is moved about through the different meridians, the media are clear and no opacities obscure vision.

The Direct Method.—Having satisfied himself of the condition of the media, the observer now brings his chair alongside the patient's, and concentrating the light upon the cornea, by means of the ophthalmoscope which he holds in close apposition with his own eye, he endeavors to approach as closely as possible to the patient's eye. If he will now relax his accommodation, by fancying that what he is now regarding is situated far in the distance, and request the patient to look straight forward into the darkness, for ophthalmoscopy should preferably be performed in a darkened room, the observer will now see the vessels of the retina and the head of the optic nerve.

Attention should be paid next to the macular region, which may be brought into view by slight movements of the patient's eye to the right or left, and finally the patient should look up, down, in and out, as requested by the observer, bringing the various parts of the fundus into view. Observations are made of the color of the fundus and of the optic disk; of changes in the elevation of the disk by focussing it and the surrounding areas; of changes in the blood-vessels, hemorrhages, pigment deposits, and degenerative areas.

The Indirect Method.—In the application of this method, the observer stations himself in a position corresponding to that assumed in the test by transmitted light and throws the light into the eye under examination by the concave mirror of the ophthalmoscope. A convex lens of +14.00 D. strength is then held before the eye to be examined, and an aerial image obtained of that portion of the fundus illuminated by the reflected light. In case the details of the fundus are not plainly seen at first, the object lens should be slightly moved to or from the eye, as a compensation for the patient's error of refraction. The image obtained by the indirect method is inverted, and it must be remembered that the upper part of the image corresponds to the lower part of the eye-ground and the right side of the image to the left of the eye-ground.

The Normal Fundus.—The normal eye presents many variations, and an extended experience is frequently necessary to distinguish congenital variations from changes wrought by disease.

The Fundus Reflex.—The color in normal eyes is bright pink or red, due to the reflection from the pigment of the retina, choroid, and the choroidal vessels. In the albino there is no fundus pigment, and the reflex is that of the choroidal and retinal vessels with the sclera as the background, and is a light pink. In the negro the reflex is gray, on account of absorption of the light rays by the abundant pigment. (See Plate III.)

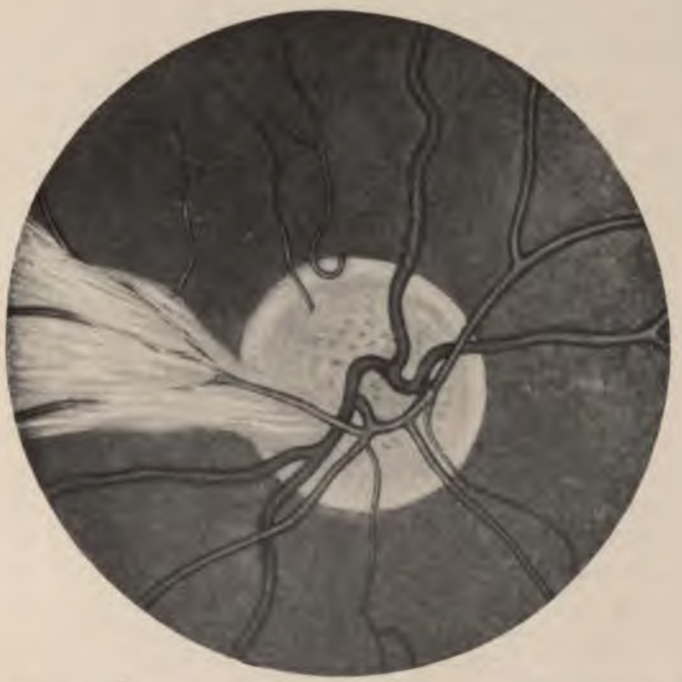


FIG. 6.—Region of optic disk showing opaque nerve-fibres and two cilioretinal vessels (from photograph).

The Papilla.—The optic disk or nerve-head is about 1.75 millimetres in diameter, appearing under the ophthalmoscope enlarged about fifteen times, and hence from 10 to 15 millimetres (one-half to six-eighths inches) in width. It is round or oval, with clear-cut margins; its centre is whitish, though the connective tissue lace-work supporting the nerve-fibres and the lamina cribosa are a mottled gray. The periphery of the disk is pink, owing to fine blood-vessels in its structure and on its surface. (Plate III.) There is usually an *excavation* in the nerve-head, due to the entrance of the retinal vessels; in some cases this is quite small, while in others it is so large that it embraces nearly the entire papilla. This excavation is physiological and should not be confused with that of glaucoma, which occupies the whole of the disk, or that which is consecutive to optic nerve atrophy.

The Blood-Vessels.—The retinal vessels leave the disk slightly to the nasal side of the centre and divide into two or more main stems,—the superior and inferior retinal, sending numerous fine branches to the adjacent retina. Just above the disk each trunk bifurcates into branches which pursue a semicircular course, curving round towards the temporal and nasal parts of the eye. The veins follow the arteries in their course more or less closely, but may be differentiated from them by being darker in color, and being about one-third larger. Both

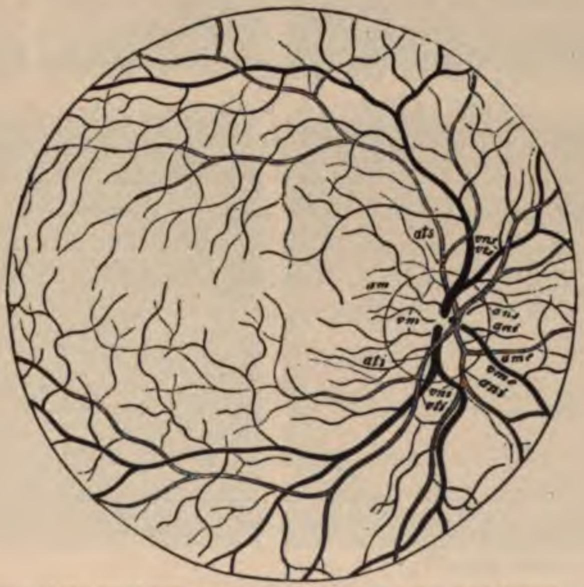


FIG. 7.—Diagram of the blood-vessels of the human retina (Leber, after Jaeger),—*ans*, *vns*, superior nasal artery and vein; *ats*, *vts*, superior temporal artery and vein; *ani*, *vni*, inferior nasal vessels; *ati*, *vti*, inferior temporal vessels; *ame*, *vme*, median vessels; *am*, *vm*, macular vessels.

arteries and veins have a light streak along the centre, which is fainter upon the veins, the *reflex streak*. A distinct *pulse* occurs physiologically in the veins upon the head of the nerve, but pulsation in the arteries is always pathological. The clinician should acquire the habit of observing the retinal blood-vessels with great care, for their condition is frequently indicative of the state of the blood-vessels elsewhere throughout the system; and on account of the possibility of viewing the actual blood-column itself an exceptional opportunity is offered of diagnosing pathological conditions of the blood.

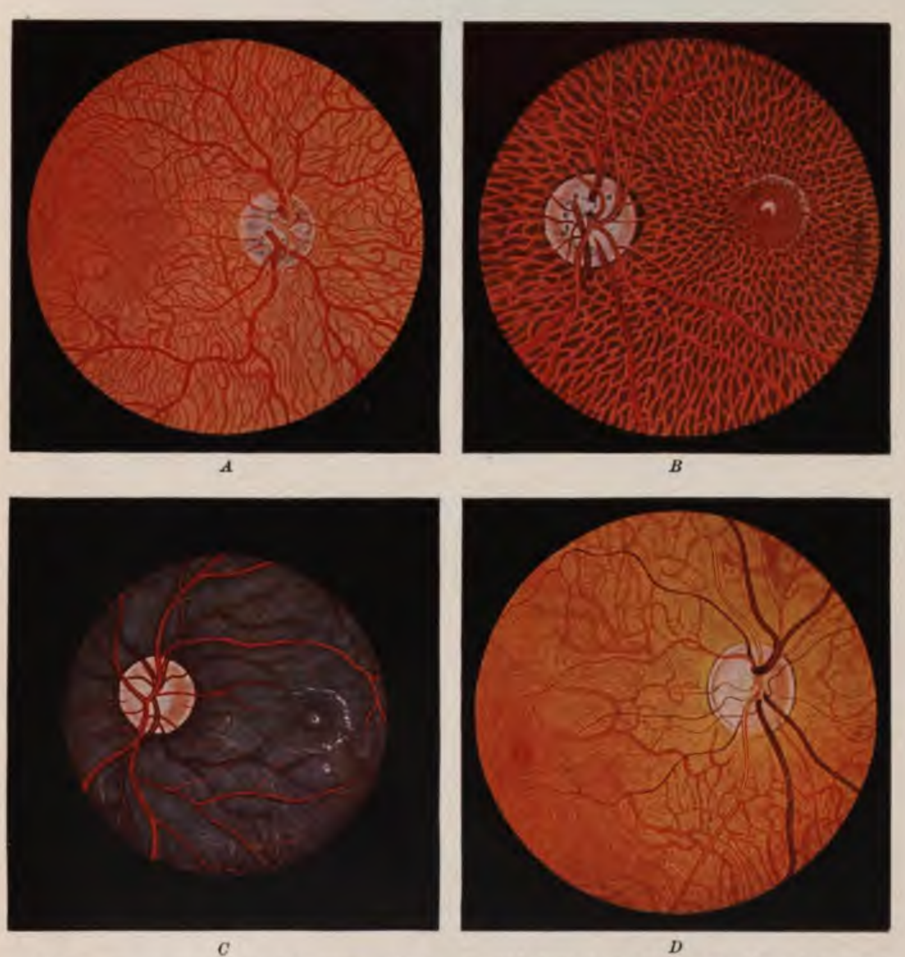
The Macula.—The yellow spot, which is situated about 2 disk diameters (3 mm.) towards the temporal side of the nerve in the horizontal meridian of the fundus, is the most sensitive part of the retina. It consists of an apparently yellow spot, surrounded by a circular or oval area, the foveal region, which is darker in color than the rest of the fundus and about the size of the disk. The centre of the macula is known as the fovea centralis, and appears as a glistening reflex. The blood-vessels do not cross the fovea but converge towards it. In order to obtain the best view of the macula, the ophthalmoscope should be slowly moved upward and inward, while the observer brings his line of vision to a point about two to two and a half disk diameters outward from the disk. When the pupil is dilated the macula may be seen by having the patient gaze directly into the sight-hole of the mirror. The periphery of the eye-ground should be carefully studied; and in order that no part of it may escape, it is well for the student to follow each branch of the central artery of the retina as far forwards as possible.

OPHTHALMOSCOPIC SIGNS OF GENERAL AND NERVOUS DISEASES.

Certain conditions of the fundus oculi are of preëminent interest and value in the diagnosis of various general and nervous diseases:

(1) Sensory affections: hyperæsthesia, anæsthesia.
(2) Vascular lesions:
    (*a*) Changes in the blood-vessels: anæmia, hyperæmia, pulsation, œdema.
    (*b*) Inflammation of the vessel-walls.
    (*c*) Hemorrhages from the vessels.
    (*d*) Stoppage of the vessels: embolism, thrombosis.
(3) Inflammation of the retina.
(4) Inflammation and œdema of the optic nerve: optic neuritis, choked disk.
(5) Atrophy of the optic nerve.

PLATE III.



Varieties of the Normal Fundus.—*A.* The albinotic fundus; albino and light blonde (after Greeff, modified by H. V. W.); *B.* The tessellated fundus; brunette (after Greeff, modified by H. V. W.); *C.* The negroid fundus; Negro (H. V. W.); *D.* The yellow fundus; Chinese (after Oeller, modified by H. V. W.)

These lesions are also described under headings descriptive of various diseases,—*i.e.*, as syphilitic, albuminuric, diabetic, and anæmic retinitis.

(1) SENSORY AFFECTIONS.

Hyperæsthesia is due to retinal irritation and hyperæmia giving rise to photophobia, asthenopia, and blepharospasm. It is usually originated by local affections, such as uncorrected ametropia, presbyopia, and muscular imbalance, but it may likewise occur in neurasthenia, gout and oxaluria. **Anæsthesia** of the retina is characterized by diminution of the central visual acuity and contraction of the visual field, or misplacement of the boundaries of the field or by changes in the boundaries for color vision (Figs. 8-9). It is a prominent symptom of hysteria and neurasthenia, is present in exhausting fevers and in convalescence, and in women with pelvic diseases. It may be induced by traumatism and by suggestion. The ophthalmoscope usually reveals hyperæmia or anæmia of the nerve-head and retina.

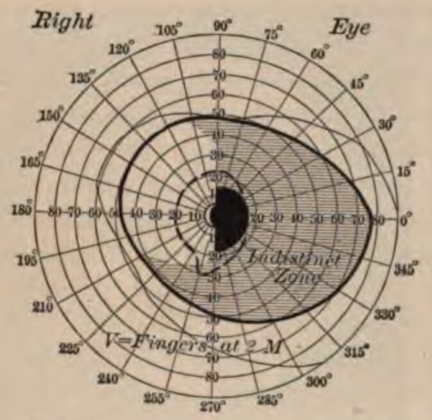


FIG. 8.—HYSTERIC FIELD, SIMULATING TEMPORAL HEMIANOPIA WITH GREAT REDUCTION OF VISUAL ACUITY.

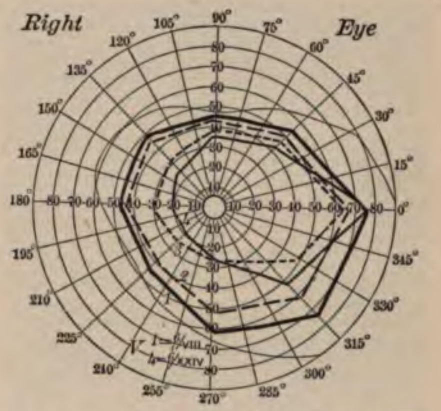


FIG. 9.—HYSTERIC FIELD, SHOWING PROGRESSIVE CONTRACTION (FATIGUE TYPE) AND OVERLAPPING OF COLOR FIELD.

1. Form limits; 2. Red, taken immediately afterwards; 3. Red, taken 5 minutes later; 4, Form, taken 5 minutes still later.

(2) VASCULAR LESIONS.

Anæmia of the disk, as well as the retina, expresses impaired nutrition. It is found in ischæmia and atrophy due to functional disturbances and blood diseases, and after severe hemorrhages. With the ophthalmoscope the arteries are seen to be apparently contracted, carry less blood, and are of a light color; the veins are full and even tortuous.

The papilla is pale, and there are, as a rule, no small vessels on its surface. (Plate IV., A.)

Hyperæmia evidences itself most prominently in the papilla, and is characterized by increase in the number of visible blood-vessels and by dilatation and tortuosity of the veins. True pathological increase of the vessels is rare, except in the beginning of optic neuritis or accompanying retinitis. The retinal vessels become abnormally vascular if the cause be mechanical obstruction to the blood, either going from or coming to the eye, the chorioid likewise shows congestion, and the pink color of the healthy fundus assumes a dark uniform red. Physiologic variations in the color of the nerve-head, on account of the contrast afforded by the varying amount of pigment in the retinal layer of the chorioid, should always be considered in making a diagnosis.

Causes.—Anæmia and hyperæmia are symptoms of both constitutional and local diseases. Among the constitutional causes may be mentioned retention in the blood of excretory products, degenerative diseases of the cerebral cortex, as epilepsy and general paralysis, hypertrophied and overacting heart from obstruction of the circulation, or thrombosis, organic disease of the abdominal organs, uterine disease, the beginning and cessation of menstruation, and finally the circulation of malarial or other toxic materials in the blood. Locally, the usual causes are chronic inflammation of the uveal tract and errors of refraction, especially hyperopia and astigmatism, with accommodative strain.

Treatment.—The treatment consists in the removal of the cause, with the use of atropine and the correction of errors of refraction, conjoined with the administration of alteratives and the stimulation of the excretory organs.

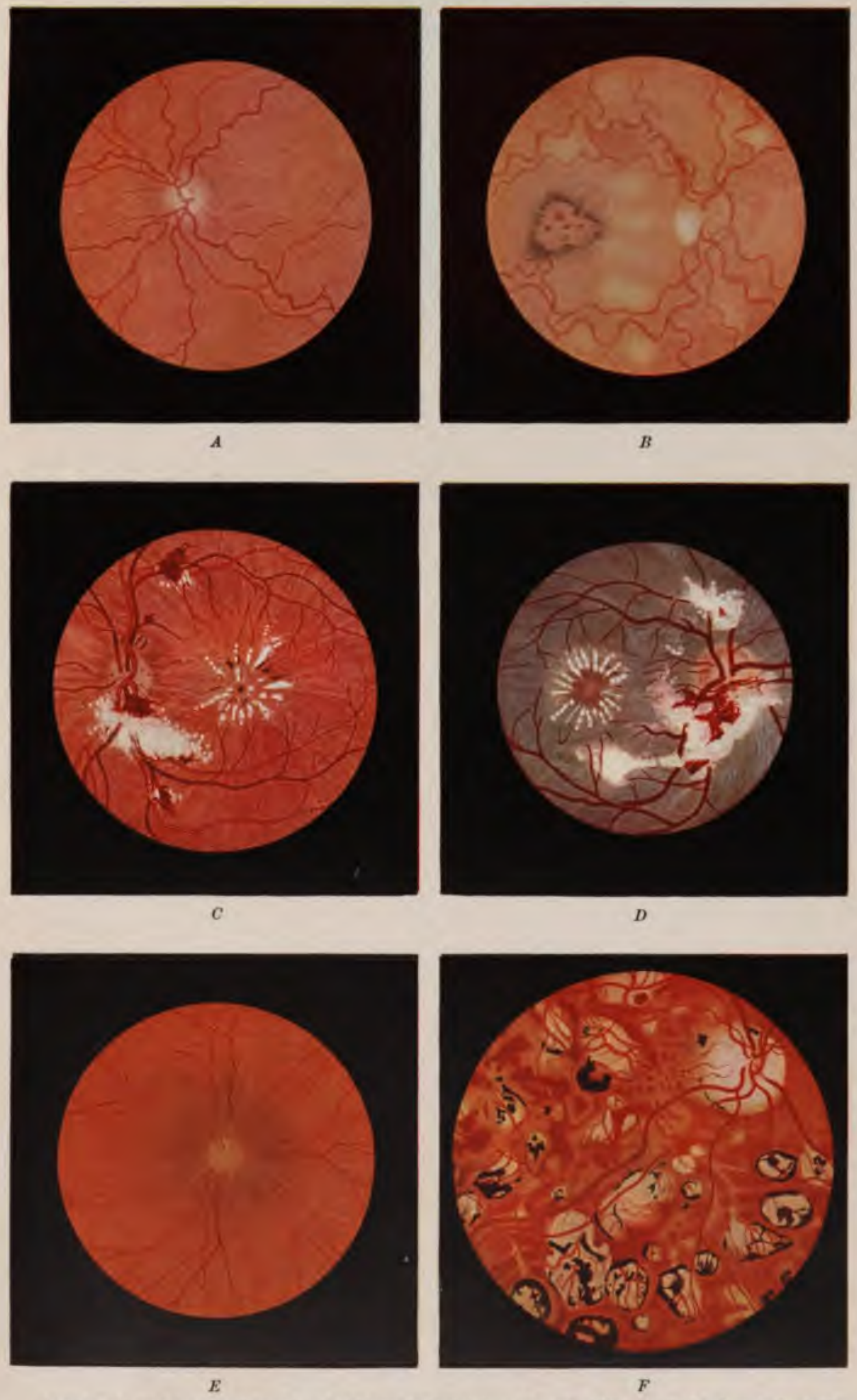
Abnormalities in the size of the retinal arteries and veins may occur as a result of some intra-ocular inflammation, or may portend some serious affection behind the eyeball.

Atrophy of the vessels occurs in association with atrophy of the retina and nerve. **Varicosity** of the veins may be congenital.

Pulsation of the retinal veins is frequently physiological, whereas pulsation of the arteries is always pathological, indicating an enlarged heart, some vascular affection of the head or orbit, or glaucoma.

Œdema of the retina may be general or limited to a certain portion. It manifests itself as an opaque, slightly elevated area, with the blood-vessels running over and through it. The vessels are decidedly dark, but lose their central light streak, and distinction between veins and arteries is difficult. In general œdema the retina is infiltrated in all its layers. (Plate IV., A and E, and V., A and B.)

PLATE IV.



Inflammations of the Retina.—*A.* Œdema in Pernicious Anæmia (Oliver); *B.* Leukæmic Retinitis (Oliver); *C.* Albuminuric Retinitis and Neuritis occurring in Pregnancy (H. V. W.); *D.* Albuminuric Retinitis in the Negro (H. V. W.); *E.* Syphilitic Retinitis (Haab); *F.* Atrophy of Retina, Nerve and Chorioid following Disseminated Chorioretinitis Luetica (Oeller).

Hemorrhages and exudation may appear in any part of the retina. Œdema is met with in syphilitic retinitis; it may be a forerunner of retinal detachment, of tumor of the retina or chorioid; it may be occasioned by traumatism, or may arise from exposure to bright light or electric flashes. Œdema of the disk occurs in the first stage of neuritis.

Inflammation of the Vessel Walls.—Vasculitis and Perivasculitis.— These conditions are, as a rule, due to arteriosclerosis or syphilis, or occur in degenerative diseases of the cerebral cortex, and are attended by other lesions in the optic nerve and retina as well as the general system. (Plate V.) (See Chapter XI.)

Perivasculitis is an inflammation of the external layers and fibrous tissues surrounding the blood-vessels. It is most commonly met with in retinitis, but may exist separately. The blood-vessels are swollen in places, but are eventually replaced by fibrous cords. (Plate V., F.)

Angeoid streaks are cicatricial changes in the retina, usually the remains of hemorrhages, though they may mark a previous detachment of the retina. They course from the disk to the periphery, are whitish in the centre, and are bordered by black lines.

Hemorrhages from the Vessels.—Apoplexy of the retina occurs most commonly in elderly people with degenerated arteries. (Plate V., E.) It is met with in cardiac hypertrophy from valvular disease, ulcerative endocarditis, chronic neuritis, gouty and uric acid diathesis, fatty and dilated heart; in scurvy, hæmophilia, purpura, and grave anæmias, malarial fever, pyæmia, leukæmia, suddenly suppressed menstruation, and the menopause.

Retinal hemorrhages may occur with or without true inflammation of the membrane. The extravasation may be small and confined to the retina, or may be large enough to fill the vitreous. Rupture of the vessels near the macula or extensive bleeding into the structure of the retina seriously interferes with vision, but many small extravasations, especially those upon the nasal side or in the periphery, may not be noticed by the patient. Hemorrhages which occur in the nerve-fibre layer of the retina are flame-shaped, while those into the deeper layers are round or oval, fresh ones appearing as dark red blotches; small hemorrhages are rapidly absorbed, leaving whitish patches surrounded by pigment, or a simple mass of pigment. If bleeding occurs into the vitreous, whitish streaks may remain, which have been given the name of *retinitis proliferans*. The distinction between simple hemorrhage into the retina and retinitis with hemorrhages cannot be definitely drawn. Most forms of retinitis are attended with effusion of blood either from diapedesis or rupture of the vessel wall. Under such con-

Unless the [illegible] small extravasations, especially marked at the posterior pole. [illegible] of the peripheral venous dilatation and arterial contraction, patches of degeneration, and pigment changes. [illegible] of the retinal extravasation takes place without apparent preceding inflammation. The masses [illegible] destruction of the retinal elements and induces inflammation in their immediate neighborhood.

Hemorrhages into the optic nerve unaccompanied by retinal extravasations are rare. They appear as irregularly outlined dark red dots, and when unaccompanied by neuritis are indicative of arteriosclerosis.

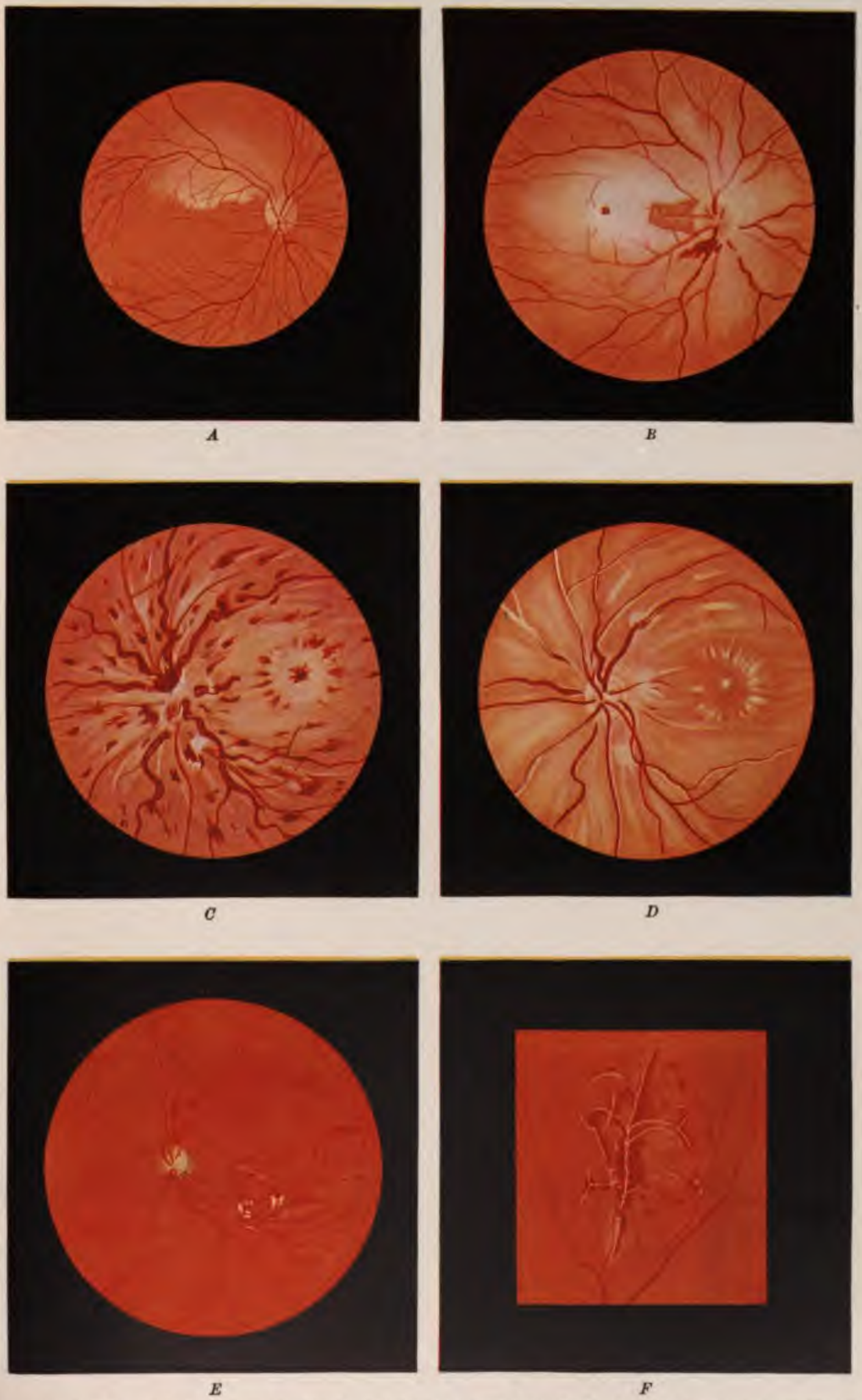
Prognosis.—Hemorrhage into the retina is a grave affection, the restoration of vision depending upon the situation of the apoplexy and degree of destruction of the retinal elements. Complete restoration of the function of [illegible] and [illegible] does not occur. Hemorrhage into the retina is a forerunner of cerebral apoplexy, a symptom of brain tumor, heart or kidney disease, diabetes, and blood affections. [illegible] a thorough investigation of the vascular system and abdominal organs should be made.

Treatment.—Although the absorption of the blood is probably as active without any treatment as with, rest of the body and eyes prevents further bleeding. Iodide of potash and small [illegible] mercurials should be administered to promote the rapid absorption of the blood elements. [illegible]

Embolism of the Central Artery.—[illegible]

Causes.—[illegible]

PLATE V.



A. Partial embolism; superior branch retinal artery (Haab); *B.* Total embolism central artery in nerve; a cilio-retinal vessel supplies a small area of the retina in which function is preserved (H. V. W.); *C.* Thrombosis of central vessels from mumps (H. V. W.); *D.* Results of thrombosis of central vessels; ıe case 6 months later (H. V. W.); *E.* Hemorrhages from retinal vessels; apoplexy of retina (Magnus); rivasculitis luetica (Magnus).

chronic nephritis is due to this cause. Syphilis and degeneration of the arteries may be suspected. Embolism has been said to occur in chorea.

Ophthalmoscopic Appearances.—These are characteristic; in complete occlusion the arteries carry no blood, and the intima only is visible; the veins and papilla are pale and the retina œdematous; sometimes small hemorrhages are found. A blood-red cherry spot appears in the fovea, resembling a small round hemorrhage. As a consequence of the ischæmia atrophy of the retinal elements and of the optic nerve ensue.

Symptoms.—In some cases the ultimate blindness is preceded by occasional transitory attacks of loss of sight. In complete obstruction of the central artery the loss of vision is immediate and entire, and in most cases permanent. In a few instances the clot has been dislodged and broken up and absorbed by treatment, securing restoration of function. In others where a part of the retina regained its function by an anastomosis through the choroidal vessels, or where cilioretinal vessels existed, a certain degree of restoration may occur.

Diagnosis.—The sudden monocular blindness, the cherry-red spot at the fovea, the diffused retinal œdema, absence of pain and inflammation, render the diagnosis positive.

Prognosis.—If the patient is seen within a few hours or days after the attack, treatment may restore vision, though if this does not occur within a short period, the retina and nerve will atrophy and the blindness persist.

Treatment.—If the case be seen within the first few days, deep massage through the closed lids by the thumb or by a mechanical masseur, or by forcibly pressing the eye back in the orbit and holding it there for a few seconds, may break up or dislodge the embolus; this conjoined with subsequent treatment by large doses of iodide of potash, saline cathartics and pilocarpine sweats has succeeded in a number of reported instances in securing partial or complete recovery.

Thrombosis of the Central Vein.—**Ophthalmoscopic Appearances.**—In this condition there is an unusual dilatation and tortuosity of the veins, the latter appearing like sausages (Plate V., C), while the arteries are contracted to narrow white lines. There are numerous hemorrhages and intense papillitis. After a few weeks the hyperæmia lessens, but atrophy of the retina and nerve ensues. (Plate V., D.)

Symptoms and Causes.—Vision, although seriously affected from the first, may not be entirely absent, though eventually it is lost. The occlusion of the vein is due to inflammation following phlebitis or endarteritis usually as a consequence of microbic infection (Fig. 10). It may occur from erysipelas, abscess of the orbit, purulent meningitis, mumps, or infectious blood-diseases. If the phlebitis has its site in a small vein, the signs are confined to the corresponding portions of the retina. It is usually limited to one eye, but cases have been reported as occurring in both.

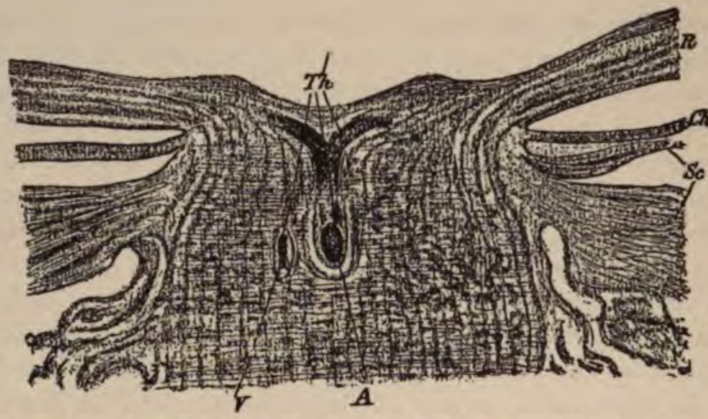


FIG. 10.—Section through optic nerve entrance in a case of thrombosis of the central retinal vein occurring in mumps.

(3) INFLAMMATION OF THE RETINA.

Retinitis may occur alone, but is usually associated with some change in the optic nerve producing neuroretinitis, with ultimate atrophy. Inflammation of the retina is announced subjectively by disturbance of vision, particularly of the central acuity, contraction or limitation of the field, scotomata, and other field disturbances. Ophthalmoscopically there may be œdema, opacity, hemorrhage, exudation, pigment changes, and alterations of the vessels of the nerve-head and retina. (Plates IV. and V.)

Visual Disturbances.—The sensibility of the retina may be increased or diminished; in the former instance there is hyperæsthesia, the retinal elements being so sensitive that subjects see best in subdued light (*nyctalopia*). In neurasthenia the retina is sometimes acutely sensitive, objects being perceived during both day and night that are not ordinarily visible to healthy eyes.

Day-Blindness.—The sensibility of the retina may be so decreased by long exposure, as in snow-blindness, that the retina cannot perceive objects illuminated by a moderate amount of light. Such disturbances may occur also from nutritional changes, as is evinced in retinitis pigmentosa and in hemeralopia (night-blindness).

Photopsia is due to irritation of the retinal cells and nerve-fibres, and is an early symptom of retinal disease. Sparks, flashes of light and color, which are more pronounced when the eyes are closed or in the dark, portend serious disease of the eye, such symptoms being commonly met with in the early stages of detachment of the retina. They may be occasioned by transient disturbances of the circulation from constitutional affections and from the exhibition of certain drugs, such as santonin, etc.

Metamorphopsia is the term applied to distortion of an object as a result of partial destruction of the rods and cones or obstruction by retinal pigment or hemorrhage. Patients with this symptom complain of overlapping of words and letters and of straight lines appearing curved or broken.

VARIETIES OF RETINITIS ASSOCIATED WITH GENERAL AND NERVOUS AFFECTIONS.

Retinitis pigmentosa is a gradual degeneration of the nerve-cells and fibres of both the retina and optic nerve with pigment infiltration into the structure of the retina. It is primarily a disease of the retina, and in some rare cases the pigment infiltration is absent. The disease begins in early life, and is inherited in 33 per cent. of the cases, 70 per cent of which occur in males. It is sometimes complicated by other congenital defects of development. About 25 per cent. of the patients are offspring of consanguineous marriages. In these families syphilis has been found in most instances. The central visual acuity is preserved to a marked extent, though peripheral vision is usually much restricted or lost. The ophthalmoscopic appearances are characteristic. The pigment cells of the retina are collected into masses, resembling bone corpuscles, and are distributed mainly along the blood-vessels, appearing first near the equator, and are usually more thickly distributed at this point. The arteries are diminished in calibre and appear as whitish lines; the veins later undergo similar changes. The retina finally atrophies and the optic nerve becomes white and very much shrunken.

Symptoms.—The earliest symptom is night-blindness (*hemeralopia*), the visual acuity being greatly lowered by diminished illumination, due to anæsthesia of the retina. Ring scotoma are sometimes present, the visual field is greatly concentrically contracted, the central vision remaining moderately acute. Nystagmus is generally present. The progress of the affection is slow, and the patients often die of intercurrent disease before vision is totally lost.

Treatment.—No restoration of vision can be secured. Strychnine injections and galvanism, and lately the high frequency faradic current, are advocated. Nerve tonics, good diet, open-air life, and iodide of potash are advised.

Syphilitic retinitis is usually secondary to chorioiditis, but the acute manifestation may first appear in the retina.

Ophthalmoscopic Appearances.—Œdema of the retina, especially near the macula, is first observed. (Plate IV., E.) This is followed by serous infiltration, the vessels become tortuous, and are partially concealed by the exudation. The retina usually retains its normal appearance towards the periphery, although isolated patches of retinitis may occur some distance from the papilla. The swelling may reach a millimetre or more, is whitish in color, and free from blood-vessels. The arteries are accompanied by white lines, indicating sclerosis of the walls and diminished calibre. The posterior part of the vitreous is infiltrated with a fine dust-like opacity. These particles of opacity are separate and distinct from one another, but may later form a membrane, due to their coalescence and incomplete absorption. Hemorrhages and pigment degeneration only occur after absorption of the œdema and the retina has been partially destroyed. A concomitant disseminated chorioiditis is likewise to be observed at this time. (Plate IV., F.)

Pathology.—As a rule, the retinitis is secondary to primary inflammation of the chorioid, although ophthalmoscopic examination may at first reveal no symptom of the latter disease. The first changes are a vasculitis, consisting of proliferation of the endothelium, infiltration of the walls of the vessels passing later into sclerosis. Subsequently infiltration of the retina from pigmentary migration occurs which extends mainly along the course of the vessels. Eventually both chorioid and retina become atrophied and degenerated, and are united in places by cicatricial changes.

Prognosis.—As syphilis is a curable disease and readily amenable to treatment, the destructive processes in the retinal and chorioidal elements are favorably influenced by prompt antisyphilitic treatment. Impairment of vision is to be expected, however, in all cases, as some degree of atrophy, not only of the retina and chorioid, but of the optic elements, always results.

Symptoms.—Central vision is diminished by the vitreous opacities and retinal œdema, and may be reduced even to the perception of light, though ultimate partial recovery is attained. The field may be contracted, and there are usually central and disseminated scotomata. Photopsia and metamorphopsia are also present. There is little pain even when iritis is present, which is a not infrequent complication, as the inflammation in the iris is usually mild and insidious and may not be recognized until after the formation of synechiæ. Though the disease is usually accompanied by a true uveitis, there is but little photophobia and but few signs of external irritation.

Treatment.—Energetic antisyphilitic treatment by mercurial inunctions should be pushed to mild ptyalism; pilocarpine should be injected hypodermically and followed by hot baths; iodide of potash should be administered in increasing and large doses, together with the application of hot compresses to the eyes. By these measures the disease may be shortened, relapses prevented, and the sight preserved.

The recurrence of retinitis in syphilis indicates a tertiary form of the disease and is an evidence of severe infection.

Albuminuric Retinitis.—Very rarely in acute inflammation of the kidney, but in about 10 per cent. of persons with chronic nephritis, pathologic degeneration of the retina and optic nerve, and with this changes in the pia mater and other brain structures arises. These inflammations of the retina, optic nerve, and meninges are due to retention in the blood of toxic elements consequent upon the disturbed functions of the kidney and impairment from deficient nutrition. Transient cerebral œdema, with increased vascular tension, which is dependent upon uræmia, is found in the late stages of nephritis and in kidney disease due to pregnancy. The latter conditions are characterized by complete and sudden loss of vision, but are unattended by retinal or optic nerve changes, whereas in the former characteristic nerve and retinal lesions are developed.

Ophthalmoscopic Appearances.—These are so characteristic that they should not be confused with changes other than those due to extensive œdema of the retina and optic nerve. In the early stages of albuminuric retinitis there is œdema and hyperæmia of the retina, the edges of the nerve are blurred and small hemorrhages occur into the retina. In the macular region there are usually small, round, or oval glistening white dots and streaks, which slowly increase in size until they form a characteristic star-shaped figure. When this appears, the veins become tortuous, the optic disk is more swollen, and more hemorrhages occur; then irregularly shaped isolated patches appear in places, which later coalesce, showing marked degeneration of the retinal elements. The hemorrhages, depending upon their location in the retina, are either round, when they are situated in the deeper layers, or flame-shaped, when they occur into the nerve-fibre layer. The whitish patches and streaks are shown by the microscope to be due to fatty degeneration of the nerve-fibres, which later become hypertrophic and œdematous. The inflammation of the retina gradually disappears after a few months or a year, and atrophy of the retina and optic nerve ensues. The disease is almost always binocular, but the ophthalmoscopic appearances vary in the two eyes, depending upon the amount of hemorrhage and fatty degeneration and the grade of the optic neuritis. (Plate IV., C and D.) It occurs only in adults and is most common in women.

Diagnosis.—The radiating macular figure is almost characteristic, although a very similar condition is seen at times in papillitis occurring in the course of tumor of the brain. Examination of the urine is necessary to establish the diagnosis.

Prognosis.—The occurrence of retinitis albuminurica is exceedingly grave; the duration of life in hospital patients averaging eighteen months, though in well cared for private patients it is considerably longer. The retinitis of pregnancy and acute nephritis, especially in young persons, is not so unfavorable. Induced premature delivery of the fœtus is usually followed by subsidence of the nephritis and a certain degree of recovery of vision, although some atrophy of the nerve with loss of central acuity and contraction of the visual field takes place, depending upon the amount of actual degeneration in the retina. It is said that this form of retinitis is usually of the serous rather than of the exudative type.

Treatment.—Treatment is that of the nephritis, or removal of the cause, as in induced abortion for the retinitis of pregnancy. Local remedies, with exception of smoked glasses and non-use of the eyes, are of no effect.

Diabetic Retinitis.—The fundus changes resemble those of albuminuric nephritis, although the degeneration is not so marked about the macula and the star-shaped figure is not usually present. Hemorrhages are more common; the optic nerve is moderately swollen; the arteries contracted; the veins dilated and tortuous. Small white patches of degeneration are usually found about the fovea, but may occur anywhere in the fundus. Retinitis in diabetes is a comparatively rare complication, being much less common than cataract, iritis, other forms of uveitis and opacities of the vitreous.

Anæmic Retinitis.—Changes in anæmia are usually limited to local anæmia, but in leucocythæmia and pernicious anæmia there are pronounced alterations. The arteries may be contracted, the veins dilated and tortuous, and hemorrhages and œdema of the retina, with a few white patches of exudation with edges colored from extravasations of blood, and slight optic neuritis, may be present. (Plate IV., B.)

(4) INFLAMMATION OF THE OPTIC NERVE.

Optic Neuritis (Papillitis, Choked Disk).—The most pronounced and important eye symptoms of certain general disorders and many forms of disease of the nervous system manifest themselves in an inflammation of the optic nerve and an associated retinitis in which fluid and solid exudation fills up the interstices of the fibres of the nerve, and even the physiologic excavation. The optic nerve elements are swollen, crowded together and distorted, thus elevating the papilla above the level of the surrounding retina, and, by pressing upon the vessels, partly obliterate and conceal them. The veins become occluded, giving rise to numerous hemorrhages. If this condition prevails for any length of time, atrophy of the nerve elements and the connective tissue takes place.

Optic neuritis may be differentiated into three classes: (1) medullary or ascending neuritis, or true inflammation of the nerve-tissue; (2) papillitis, or simple swelling of the intra-ocular portion of the nerve from irritation or pressure; (3) descending neuritis, originating from intracranial diseases, affecting the nerve or its sheaths.

Ascending neuritis commences in the papilla and usually terminates in the brain. **Descending neuritis** commences in the brain, later finding its way down the nerve-sheath or nerve elements into the eyeball. **Choked disk** is an arbitrary expression, denoting swelling of the papilla from 2 D to 6 D, or even more. (The difference between the highest point on the disk and that of the surrounding retina in diopters may likewise be expressed in millimetres, 2½ D being equivalent to 1 millimetre of swelling).

Causes.—The causes of optic neuritis are ocular, orbital, intracranial, and constitutional.

Ocular causes include chorioiditis and retinitis, which are, however, usually dependent upon some constitutional cause.

Orbital.—Fracture of the orbit, cellulitis of the orbit, hemorrhage into the cellular tissue of the orbit or nerve-sheaths, deformities of the skull, affections of the neighboring sinuses, etc., may be enumerated under this heading.

Intracranial.—Optic neuritis may occur in the course of diseases of the brain; these may be enumerated in order of frequency, as follows: meningitis, tumors, gummata, sclerosis, tubercle, cysts, and aneurism. A low grade of retinitis and optic neuritis is seen in some cases of epilepsy and general paralysis. In children probably the most frequent cause of neuritis is tubercular meningitis. Intracranial affections give rise to optic neuritis by direct pressure upon the chiasm, the optic nerves, and the tracts; or by occasioning increase of the ventricular and subarachnoid fluids and disturbing the equilibrium of the different portions of the brain; or by disturbances of nutrition through interference with the circulation.

The precise manner in which intracranial growths occasion optic neuritis is still disputed. Von Graefe was of the opinion that the neuritis was due to a strangulation of the lymphatics of the head of the nerve, as a consequence of the increase in intracranial pressure, occasioning a blocking of the cavernous sinus and of the tributary ophthalmic vein. This theory has been supported by Manz and Schmidt-Rimpler, who demonstrated that the fluid displaced by a cerebral tumor found its way into the lymph-spaces of the optic nerve, distending the intersheath spaces with fluid. Leber and Deutschmann, on the other hand, believe that the choked disk is not merely a stasis but claim that the presence of the fluid sets up a toxic condition which gives rise to a true inflammation of the nerve. Others incline to the view that the neuritis is a true descending one, as they have shown that there is an increased cellular exudation in the tissues surrounding the cerebral tumor, which extends the whole way from the tumor to the nerve and along the latter to the eye. In view of these facts, it would appear that the element of intracranial and intervaginal pressure plays an important part in the production of choked disk, but that the latter, in the majority of cases at least, depends upon an active inflammation.

Choked disk and optic neuritis occur in about 90 per cent. of cases of brain tumors, irrespective of their size or location. The absence of optic neuritis does not exclude the diagnosis of brain tumor, but its presence is a strong confirmatory sign. Tumors of the occipital region and the base of the brain are apparently more often accompanied by choked disk than those in other portions (Compare Chapter IX).

Constitutional.—Optic neuritis sometimes occurs in eruptive fevers, diphtheria, epidemic influenza, and other contagious diseases; it may also be occasioned by albuminuria, especially of the puerperal type, diabetes, leucocythemia, toxemia, pernicious anæmia, acute rheumatism after exposure to cold, and menstrual affections.

It is believed by some that in all cases of optic nerve atrophy there is first an active, although evanescent, primary inflammation of the optic nerve, which is manifest though for a short time upon the optic disk. In spinal cord affections a slight degree of neuritis or neuroretinitis is a forerunner of optic atrophy. Optic neuritis occurring in the course of syphilis is an evidence of the tertiary stage of the disease.

Ophthalmoscopic Signs.—The inflammatory process, as observed by the ophthalmoscope, may be arbitrarily divided into three stages, which pass imperceptibly into each other.

First Stage.—The border of the papilla is blurred by infiltration or swelling; the physiologic excavation is filled in and the disk reddened, as a consequence of relative increase in the size of the blood-vessels, whereby fine twigs invisible in health are brought into view, and the veins increased in size and tortuosity. The main arteries of the disk are contracted, the connective tissue becomes hypertrophied and opaque and conceals the disk outlines. The papilla projects forward, as may be determined by focussing the blood-vessels and its surface. (Plate VI., E.) During this stage there are no marked changes in the retina excepting dilatation, tortuosity and dark color of the veins, and the disease may not advance ophthalmoscopically beyond this condition.

Second Stage.—If the inflammatory process continues in intensity, these ophthalmoscopic signs increase. Then the swelling of the nerve may become more marked and the disk may project as much as three millimetres (7½ D) into the vitreous. (Plate VI., F.) Its outlines are hidden by exudation and hemorrhage, and the swelling overlapping in all directions apparently doubles the size of the papilla. Its position is thus only determined by the entrance of the blood-vessels which are obscured and interrupted in places and difficult to follow. The veins are enormously dilated, tortuous and dark in color, apparently outnumbering the arteries, which are proportionately reduced in number and calibre and carry little blood, showing in places as simple white lines. Small flame-shaped hemorrhages appear near the disk. The retina in the neighborhood is œdematous, and its fibre layer hypertrophied, showing radiating white lines. This stage varies in duration from a few weeks to several months, gradually passing into the stage of degeneration.

Third Stage.—Absorption of the blood and exudation now takes place, the swelling of the nerve-head declines, the periphery contracts, the exudative material is absorbed or transformed into connective tissue, the veins are less tortuous, and the arteries smaller. The papilla becomes a dirty grayish white, and a firm, organized exudation fills in the physiologic excavation, completely hiding the lamina. The nerve passes ultimately into complete atrophy. (Plate VI., A.)

Symptoms.—*Visual Acuity.*—The vision is usually but little affected at first, and thus few patients are seen until the second stage ensues, when the vision is usually somewhat reduced, the reduction becoming gradually worse during the continuance of the disease. Large hemorrhages in the region of the macula may, however, cause more or less sudden blindness. Exceptionally there is no great deterioration of vision until the third stage, and thus a considerable degree of papillitis may exist with normal visual acuity. Hemorrhage or exudation into the macular region and other parts of the retina, permanently diminish the function of the retinal cells and fibres by pressure, and later by cicatricial tissue formation and contraction. As a rule, vision diminishes to light perception or ultimate blindness in the cases which outlive the ocular affection.

Visual Field.—The changes in the visual field are important in localization of the cerebral lesion. The most common defects are

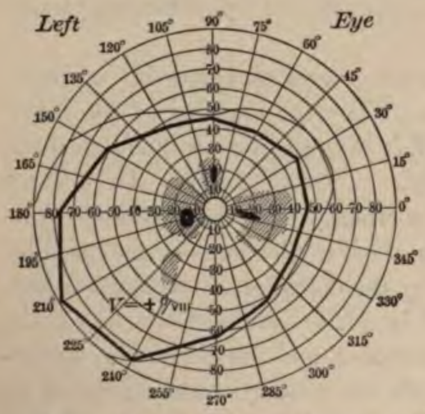


FIG. 11.—Enlarged blind spot with paracentral absolute and relative scotomata in optic neuritis from brain tumor.

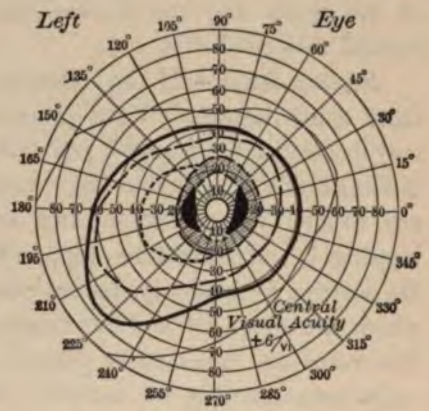


FIG. 12.—Pericentral absolute and Ring scotoma for colors, in syphilis, due to probable gumma of optic nerve.

peripheral contraction, enlarged blind spot, or central scotomata. The former is characteristic of uniform pressure upon the nerve by fluids or solids in or external to its sheath, while the latter indicates axial neuritis with loss of function of the central nerve-fibres. Sector-like defects involving definite portion of the visual field, such as hemianopsia, which may be,—*i.e.*, homonymous (nasal or temporal), heteronymous (or lateral) tetranopsia (loss of a quarter of the field), and smaller defects occur, which are symptoms of descending optic neuritis, due to lesions of the chiasm, tract or deep centres. These are usually binocular and symmetrical. Monocular defects arise from pressure upon the nerve in the orbital canal or orbit, or upon a portion of the asm or upon small areas of the occipital lobes.

The color fields diminish more rapidly than the perception for white and form, though there is usually peripheral contraction for all colors. As the disease progresses, green is first lost, then red, and finally blue. The loss of the blue field is contemporaneous with marked contraction of the form field and indicates advancing atrophy. As the scotomata and sector-like defects in the visual field, which occur from intra-ocular disease secondary to changes in the retina, frequently resemble those from changes in the cerebral centres, it is necessary to rely upon ophthalmoscopic examination to establish the diagnosis. (See Chapter IV.)

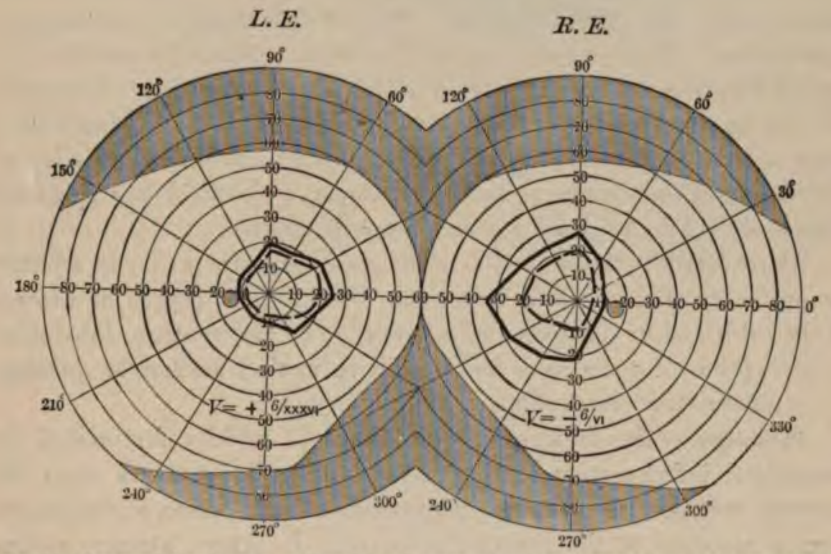


FIG. 13.—Temporal hemianopia and peripheral contraction from tumor of optic chiasm, causing bilateral choked disk.

Prognosis.—Optic neuritis of all grades is grave, because the intracranial and constitutional diseases of which it is a lesion involve essential organs of the body. An unfavorable prognosis as to sight may usually be given, with the exception of cases of toxic amblyopia, in which full recovery often occurs after removal of the cause. Atrophy invariably follows the inflammation, and as the degeneration of the nerve progresses, vision diminishes. The atrophy generally advances more rapidly in one nerve than the other, so that some useful vision may be preserved in one eye even after the other has become blind. As regards life, the occurrence of an optic neuritis often indicates grave cerebral involvement, and when it is concomitant with retinitis or advanced renal or vascular lesions, its occurrence is usually prognostic of a lethal ending within a few weeks to two or three years, at most, depending upon the gravity of the central or constitutional lesion.

Treatment.—The treatment depends upon the cause, for which a complete physical examination is essential. Discovery of albumin, casts, or sugar in the urine, determines the line of treatment, which, as a rule, is to be given by the family physician. If the case is found to be of cerebral origin, the oculist may be seen only in consultation. Syphilitic cases should be combated by energetic mercurial inunctions, large doses of iodide of potash, pilocarpine sweats. The rheumatic, albuminuric, and toxic types are likewise benefited by pilocarpine. Acute forms, where exposure to cold has been followed by neuritis and loss of vision, are helped by blood-letting and diaphoresis. Salicylate of soda in doses corresponding to the weight of the individual,—*i.e.*, 1 grain for each pound of weight a day,—are of service. Regulation of the menses is an important factor in women. Ethmoid and sphenoid disease should be searched for and properly treated.

Acute Retrobulbar Neuritis.—The optic nerve-fibres may be affected primarily by disease posterior to the eyeball from injury or disease of the orbit and of the accessory cavities, or by rheumatism, inhalation or absorption of toxic substances, such as methyl alcohol or quinine, or by influenza, acute alcoholism, and autotoxæmia.

Pathology.—The function of the nerve is rapidly diminished by interstitial inflammation of the nerve-fibres, or by pressure upon the nerve or vessels. In some cases the inflammation subsides, as the exudation is absorbed or the pressure removed. In others, atrophy ensues with more or less complete loss of vision.

Symptoms.—There is rapid loss of visual acuity, perception of light in some cases being lost in a few hours or days. The visual field may be peripherally contracted, but, as a rule, there is a central scotoma which increases in size until only a small rim of peripheral field remains. The central scotoma is at first only for color,—particularly for red and green. Partial recovery occurs, followed later by further loss of vision. Weakness or paralysis of the facial or other nerves may accompany or precede the optic nerve affection.

Ophthalmoscopic Appearances.—The ophthalmoscope shows the nerve-head to be hyperæmic with discolored surface and obscured edges; the arteries are contracted, and the veins dilated, and in rare cases there are a few small hemorrhages.

Prognosis.—Recovery of useful vision follows early energetic treatment, though cases usually go on to more or less complete blindness.

Treatment.—Removal of the cause with local blood-letting, large doses of salicylates in rheumatic cases, strychnine hypodermically in increasing doses, iodide of potassium, and mercurial inunctions are indicated. Pilocarpine sweats eliminate poison and assist the action of the internal remedies.

Chronic Retrobulbar Neuritis (Toxic Amblyopia).—The optic nerve may degenerate from the constant absorption of poisons, notably large quantities of tobacco or alcohol, or, as is usually the case, their combined action. Numerous other poisons,—*i.e.*, lead, quinine, the methyl compounds, salicylic acid, napthalin, nitrobenzol,—will produce a similar inflammation. Toxic amblyopia may also arise in the course of some constitutional diseases, such as diabetes, and one of the theories of choked disk is that it is developed by a local toxemia arising in the brain.

Pathology.—The disease consists in a true axial medullary neuritis, possibly secondary to degeneration of the macular ganglion cells, although it is more probable that it arises in the bundle of the nerve-fibres supplying the macular region. It is a gradual degenerative process and which, unless checked, encroaches upon the other fibres adjoining the papillomacular bundle until the entire nerve becomes atrophic.

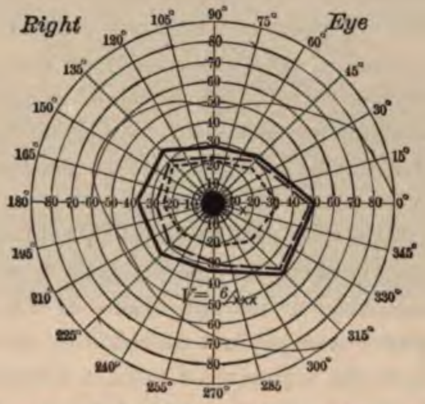


FIG. 14.—Toxic amblyopia; small absolute central scotoma; larger central scotoma for red and green; contracted field.

Symptoms.—The impairment of vision is gradual, and consists in blurring of sight, which is not relieved by glasses. The pupillary reflex may be disorganized so that the pupil dilates with the accommodation. The visual acuity varies from slight derangement to entire loss of central acuity, depending upon the amount of central scotoma. As a rule, there is more disturbance for color than for form, the characteristic signs of the disease being central scotoma for colors, more particularly for green and red. Later in the disease the scotoma, which was at first relative, becomes absolute, increases in size, and finally the peripheral field becomes concentrically contracted.

Ophthalmoscopic Appearances.—As a rule nothing distinctive can be seen in commencing cases, with the exception that the outline of the nerve is not so sharp and clear as in the normal subject (Plate V., B.), but in advanced cases the nerve-head is usually pale in the lower temporal quadrant,—*i.e.*, in the region occupied by the macular fibres.

Prognosis.—Prompt treatment preserves the visual acuity, and in favorable cases vision may be entirely restored, the amount depending upon the degree of destruction of the nerve-fibres.

Treatment.—Removal of the cause. If this be alcohol or tobacco or both, the patient may have to receive special general treatment, which can usually only be properly carried out away from his usual surroundings, in a sanitarium or hospital. Increasing doses of strychnine, hypodermically, for courses of several weeks, with intermissions of a week or two, may restore much of the lost vision. Nitroglycerin and nitrite of amyl may be used. If there be exudation into the nerve, the iodides are of value. In anæmia, the employment of iron and other tonics, nourishing diet, exercise out of doors, early hours, and plenty of sleep, are necessary. Galvanic electricity and the high frequency faradic current seem to stimulate the retinal elements to renewed activity.

(5) OPTIC NERVE ATROPHY.

Atrophy of the optic nerve may be primary or secondary, the types being readily distinguished by the appearance of the optic disk.

Primary Atrophy.—Atrophy not preceded by pronounced papillitis or neuroretinitis is, in the majority of cases, a symptom of spinal cord disease, usually of sclerosis of the posterior columns (locomotor ataxia). It occurs less frequently in disseminated sclerosis, lateral sclerosis, epilepsy, and general paralysis. Toxic amblyopia, both acute and chronic, the specific fevers, diabetes, and arteriosclerosis also lead to atrophy of the medullary fibres. There is likewise an hereditary form following retrobulbar neuritis. It is a mooted question whether or not œdema of the nerve is not always present and capable of being observed ophthalmoscopically in the early stages. Where it is concomitant with spinal disease the structural changes in the optic nerve may apparently precede the spinal symptoms for months or years, and thus the diminishing of the visual function may be the first symptom of the general affection. (See Chapter XII.)

Anatomical Changes in Primary Nerve Atrophy.—*Ophthalmoscopic Appearances.*—The papilla gradually assumes a chalky-white appear-

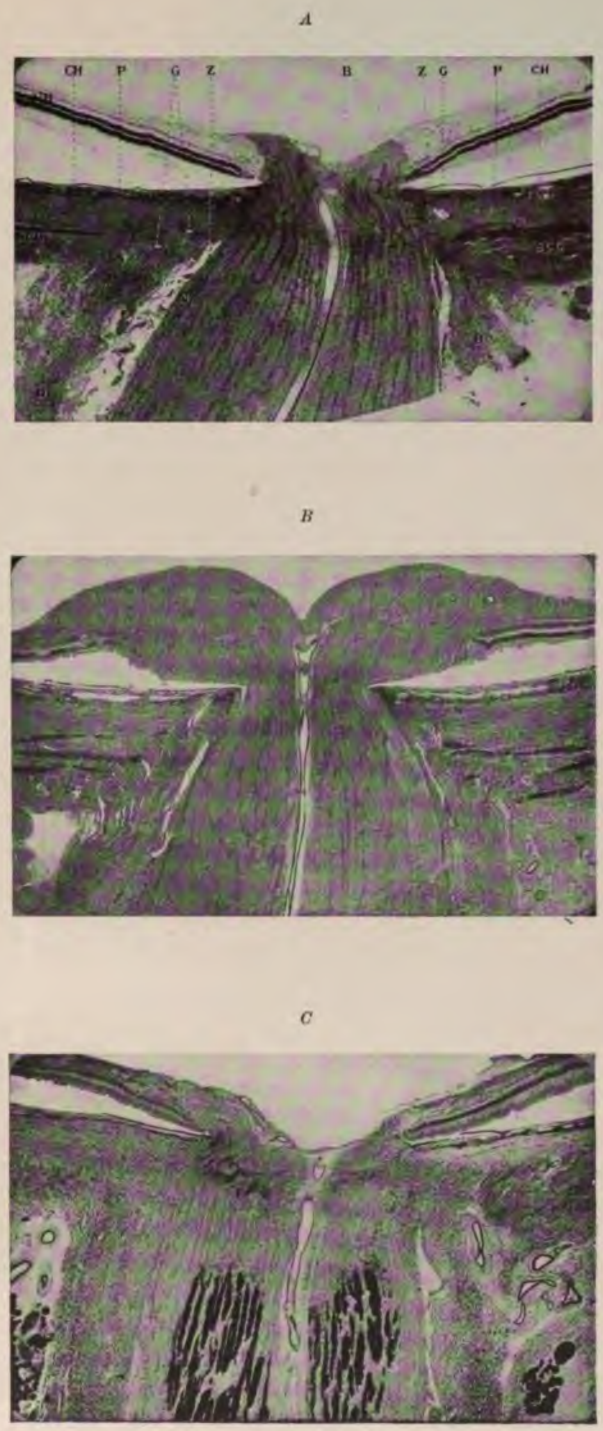


FIG. 15.—Sections through the optic nerve. *A*, normal nerve; *B*, papillitis; *C*, optic atrophy. (Reproduced from Elschnig.)

ance or it may acquire a bluish-green tinge. As the atrophy progresses, contraction of the nerve-fibre bundles and their surrounding elastic tissue takes place, causing cupping of the nerve. The cupping is gradual and occupies the surface of the nerve. All the blood-vessels become contracted and the arteries may be entirely obliterated, being replaced by white streaks, especially when the disease is due to arteriosclerosis. This process causes first a flattening of the central nerve-fibres with circumscribed atrophy of the marginal or central nerve-fibre bundles, and the changes in the papilla are always preceded by failure of vision and defects in the visual field. Atrophy of the retina follows that of the nerve.

Microscopic Appearances.—This is not an ascending atrophic process, and the nerve does not become as thin and attenuated as in the neuritic inflammation. On section, the septa are found to be well preserved, and the fibres transformed into fine fibrillæ which stain with carmine. The blood-vessel walls are sclerosed, but the septa only become thickened quite late in the disease. Some of the nerve-fibres remain healthy, and cases may escape the sclerosis entirely. The surface fibres are the first to degenerate and this may show in the visual field as peripheral alterations. (Fig. 15, A. B. C.)

Subjective Signs.—The acuity of vision depends upon the degree of degeneration of the nerve-fibres. The first symptoms are diminution in the light sense and contraction in the color fields; the patient demands strong light and has difficulty in getting about in the dusk or dark. The light sense may be tested by determining the acuity of vision in different degrees of illumination, or by the Förster photometer.

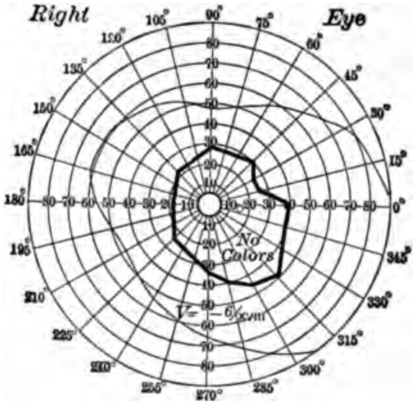


FIG. 16.—Optic nerve atrophy in tabes dorsalis.

Usually the contraction of the color fields is greater than that for white, and a large white field may remain when the color vision is entirely gone (Fig. 16).

Prognosis.—The disease terminates in partial or complete blindness.

Treatment.—Treatment is that of the constitutional or cerebral disease. Electricity is much used, but is of little avail. Nitroglycerin and nitrite of amyl and hypodermic injections of strychnine temporarily improve vision. Administration of the iodides of mercury, potash, soda, and strontium, and the bromides and iodides of gold, mercury, and arsenic, and sweat baths are recommended.

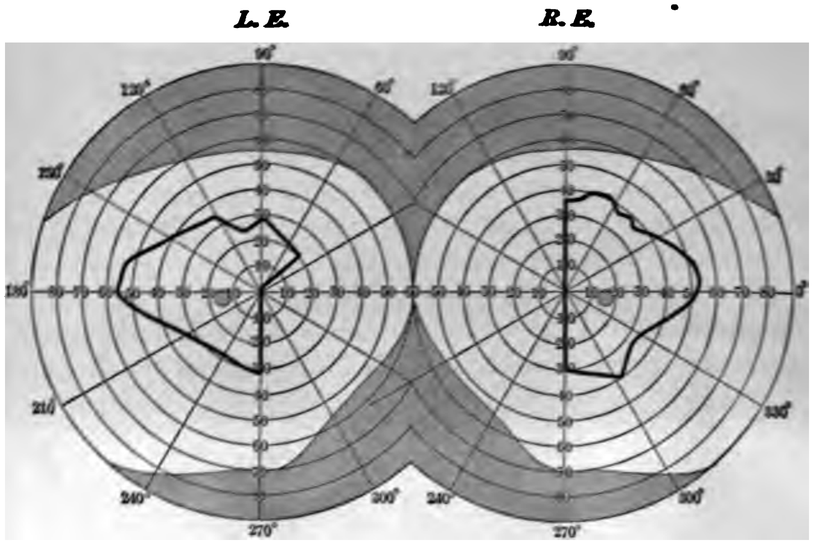


FIG. 17.—Nasal hemianopia. (After Veasey.)

Secondary, Ascending, Consecutive, or Neuritic Atrophy.—This condition is the result of inflammation of the orbital end of the optic nerve following retinitis, neuroretinitis, choked disk, or chorioretinitis. The inflammation may furthermore be induced by intracranial lesions, tumors, syphilis, encephalitis, meningitis, or distention of the third ventricle pressing upon the chiasm, or may result from retrobulbar neuritis, injury, œdema, or pressure on the optic nerve at the optic foramen, from alcoholism, from the manifestations of syphilis, tuberculosis, nephritis, etc.

Ophthalmoscopic Appearances.—The changes in the optic nerve are somewhat different from those of primary or spinal atrophy. Thus the nerve-head is usually of a dirty-gray color, is not cupped, and the edges are not so well defined as those of spinal atrophy. (Plate VI., A.)

Microscopic examination demonstrates that this is only a strictly ascending process when following purely intra-ocular inflammations, as chorioretinitis, for in most cases anatomic examination shows that choked disk or optic neuritis involves the nerve behind the globe, even as far back as the optic chiasm and intracranial divisions.

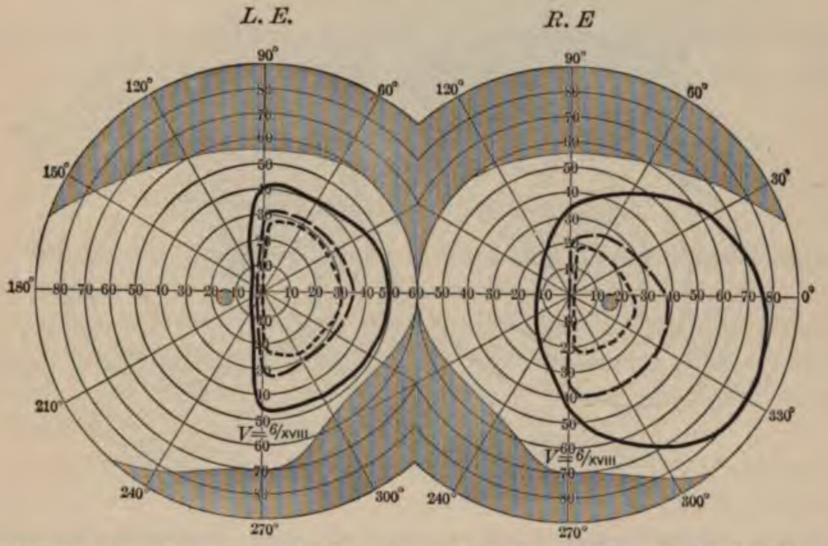


Fig. 18.—Homonymous hemianopia (lateral) with external ophthalmoplegia occurring in disseminated sclerosis with nuclear paralysis.

The subjective signs are much the same as in spinal atrophy. There is a loss of vision and contraction of the visual field, the various changes in the field depending upon the intracranial lesion or the amount of retina that has deteriorated as a result of the intra-ocular disease.

Treatment.—Therapeutics may check the advance of the atrophy and postpone the inevitable blindness, as has been noted in discussing primary atrophy.

Hereditary optic-nerve atrophy is a sufficiently peculiar form to demand separate description. Subjects of this form of atrophy are usually affected at puberty or early manhood. As a rule, their skulls are tower-shaped, and belong to the so-called scapho- and dolicho-cephalic varieties. The sphenoid bone is not fully developed until rather late in life; it contains the optic foramen, and any irregularity in its growth may cause in this type of skull a lateral pressure which occasions the peculiar ingrowing visual field defect which invades the field of fixation and ultimates in the loss of central vision. (See Chapter XIV.)

In some cases the central scotoma is not accompanied by marked narrowing. This may be explained by the fact that the axial fibres immediately behind the bulb are quite lateral, and may continue so to the foramen.

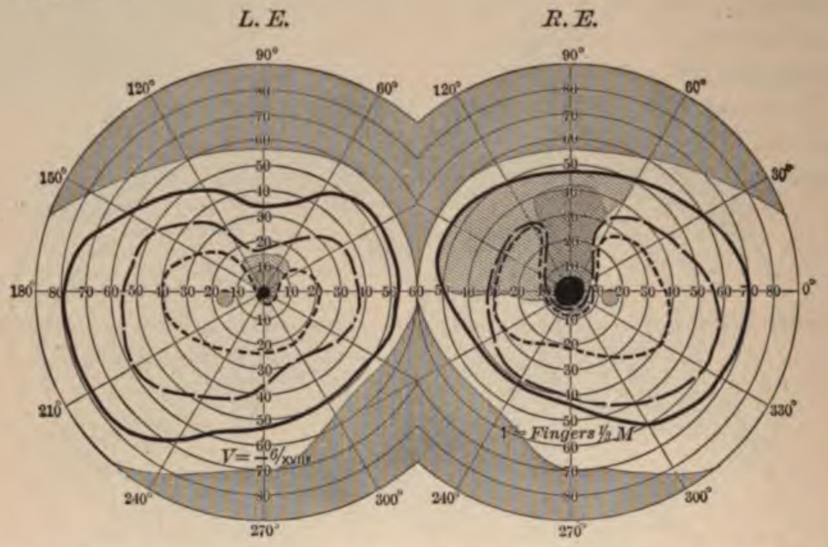


FIG. 19.—Hereditary optic-nerve atrophy, showing development of central absolute and relative scotomata.

Cases of this disease usually occur in young males and are transmitted from one generation to another through the females, who escape.

Treatment is the same as for other forms of atrophy, but is practically unavailing.

CHAPTER IX.

TUMORS AND OTHER LESIONS OF THE BRAIN, ESPECIALLY THOSE IN RELATION TO THE VISUAL APPARATUS AND OCULAR NERVES.

BY WILLIAM G. SPILLER, M.D.

GENERAL SYMPTOMS OF TUMOR.

So much has been written on encephalic tumors, both by American and foreign writers, that it has seemed well in this chapter to pay more attention to new growths that are of special interest to ophthalmologists. American writers have made notable contributions to this subject. Tumors not connected with the visual system will therefore be treated rather briefly in this chapter.

Tumors of the brain may cause symptoms that are not indicative of the position of the growth but merely show that something abnormal is developing within the cranial cavity producing an increase of intracranial pressure. The most important of these symptoms are headache, often persistent and with exacerbations, nausea and vomiting, general convulsions, vertigo, and choked disks. They do not imply necessarily that a tumor is growing within the brain, and they may be caused by meningitis, abscess, or other lesions. There are in most cases of tumor certain signs and symptoms that give some clue to the location of the cerebral growth; and the determination of these is of great importance. Choked disk in itself does not show whether a tumor is in the cerebrum or cerebellum. Marcus Gunn's opinion that unilateral, or preponderatingly unilateral optic neuritis is in favor of the tumor being on the same side as that on which the neuritis is more intense can not be accepted for all cases. Purves Stewart, for example, says he has seen a case of large frontal tumor in which the optic neuritis was both earlier and more intense on the opposite side from the lesion. This is an interesting observation, because we might expect that if unilateral optic neuritis were indicative of the side on which the tumor is situated, it would be so especially when the tumor is in the frontal lobe.

LESIONS OF THE OCCIPITAL LOBE.

There are certain signs and symptoms pointing to tumor of the occipital lobe, and yet they are not pathognomonic. Early developing hemianopsia or hemiachromatopsia, sensations of light, visual hallucinations in the blind fields, with absence of Wernicke's hemianopic pupillary sign and with the general symptoms of brain tumor are indicative of a growth in the occipital lobe. In some cases the symptoms of cerebellar tumor are added and are caused by pressure upon the cerebellum. (*For remarks on optic neuritis see* Chapter VIII, p. 375.)

Hallucinations of Sight.—Ferrier and Jackson are said to have been the first to observe photopsia in disease of the occipital lobe. The visual hallucinations usually appear in the blind fields. Wilbrandt and Henschen believe that these visual hallucinations occur only with subcortical hemianopsia, and not when the cortical visual centre alone is disturbed. (*See also* Chapter IV, p. 174.)

Harris, in referring to Swanzy's view that hallucinations of sight in the blind fields are possible in lesions of the optic radiations but not in hemianopsia the result of cortical damage, says that the reverse seems to be more common. The only case with necropsy supporting Swanzy's view is that reported by de Schweinitz. Harris refers to a number of cases of visual hallucination with hemianopsia but almost all were without necropsy.

Oppenheim mentions that visual hallucinations and illusions may be caused by intraocular lesions, and refers to Uhthoff. This phenomenon I also have observed. I recall a patient under the care of Dr. C. K. Mills whom I saw with him. The man had hallucinations of sight, and saw various objects, especially horses, as a symptom of hemorrhage into the retina.

L. Bruns believes that visual irritation symptoms preceding or accompanying hemianopsia are of value as indicating a location of the lesion in the occipital lobe. These symptoms may be spots or lines of light before the eyes, or hallucinations of sight.

In a case observed by Byrom Bramwell [1] in which numerous tumors were found in the brain, and the occipital lobes and each optic thalamus were implicated by the tumors, the patient had frequent paroxysmal attacks lasting about five minutes at a time, with headache, flushing, and flashes of light in the eyes. The light was especially bright in the right eye and had the brilliancy of electricity, and varied in size from a button to a large globe. During these paroxysms the patient was completely blind. Hemianopsia did not appear to be present after an attack. Bramwell attributes these flashes of light in the right eye to irritation of the left half-vision centre. It is well to remember, however, that each optic thalamus was the seat of tumor, and it is possible that the irritation symptoms were caused by the implication of these bodies; such a complication was not present in another case observed by him.

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[1] Byrom Bramwell, Brain, 1899, vol. xxii. p. 41.

In this second case the cicatrix of an old syphilitic gumma was found on the outer surface of the posterior end of the left occipital lobe. The patient, a man, complained of severe headache confined to the back and top of the left side of the head. He had peculiar sensory (visual) Jacksonian epilepsy. The attacks occurred on an average about once a year, and each attack, consisting of numerous paroxysms, usually lasted three or four weeks. The paroxysms occurred every five or ten minutes, and lasted from half a minute to three minutes. They consisted of flashes of light in the right eye, and the light was said to be red and white. For some days after the attack the patient was unable to see to the right side (right-sided homonymous hemianopsia).

Jolly has shown that scotoma scintillans may result from a disturbance of the visual tracts, and more recently A. Pick[2] has demonstrated by the report of four clinical cases that hallucinations of sight in the hemianopic fields may be caused in the same way. The first of his cases shows that a focus of softening probably existed in the left angular gyrus. Important symptoms in this case were paraphasia and paragraphia and persistent right lateral hemianopsia. At least on one occasion everything in the right side of the fields of vision was of a reddish color and changed later to green. For about half an hour a dog was seen by the patient walking along with him at his right side. This was merely a visual hallucination.

It seems that hallucinations of sight may be caused by disturbance of any part of the visual system from the eyeball to the cortical layer of the occipital lobe.

Pick has observed bitemporal scotoma scintillans, and he attributes this phenomenon to a lesion of the chiasm.

According to Uhthoff,[3] disease of the retrobulbar and intracranial optic pathways relatively seldom causes visual hallucinations, but it did in one of his cases and he refers to some others in the literature (Christian, de Schweinitz).

Harris reports a case of partial hemianopsia in which a peripheral zone of the left lower quadrant remained intact. The lesion was believed to be a softening of the right cuneus. The patient had frequent attacks in which he saw red and green lights always to the left in the blind field, and they appeared to be moving in both directions, from the periphery towards the centre and in the reverse direction.

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[2] A. Pick, Amer. Journ. of the Med. Sci., January, 1904, p. 82.

[3] Uhthoff, Monatsschrift für Psychiatrie und Neurologie, vol. v.

I know of a case in which the diagnosis of fracture of the base of the skull was made. The patient at times had visual and olfactory hallucinations and saw his bed and other objects move about him and turn upside down.

In the case reported by de Schweinitz the patient had visions of chairs, tables, and other articles of furniture, not really present, in the fields which afterward were obliterated. Left lateral hemianopsia developed, the dividing line passing in advance of the fixation-point. The patient was unable to say whether the visual hallucinations disappeared immediately after the hemianopsia set in, or whether they remained for some time in the dark half-fields. At the necropsy made by Dercum gummatous infiltration at the base of the brain pressing upon the right optic tract was found. De Schweinitz[4] says he was not aware that any lesion of the occipital lobe was present, and it was not probable from the symptoms that one would be found.

Bregman[5] has described green and violet vision occurring in tabes dorsalis with optic atrophy, and probably resulting from the irritation of the optic nerves. Alter observed a case of paretic dementia in which attacks of green vision, lasting one or two hours, occurred, and occasionally were followed by achromatopsia. Objects were seen in various shades of green. It seems probable that these peculiar visual disturbances in which objects appear red or green may be caused by irritation of any part of the visual system, and it is not mere chance that dictates the colors seen. The fibres conveying sensation of red and green, if such fibres exist, seem to be chiefly affected in optic atrophy, and therefore from irritation sensations of light might be more likely to appear as red and green vision. The violet seen by Bregman's patient contained red. We should, therefore, from a study of the cases mentioned, be very careful before assuming that subjective disturbances of color vision indicate a lesion of the occipital lobe.

I am inclined to think that inverted vision, or mirror vision, may be caused by lesions of the occipital lobes, but much study is needed in regard to this phenomenon.

Alexia and Optic Aphasia.—More valuable are alexia and optic aphasia as signs of a lesion of the left occipital lobe. These signs are explained by L. Bruns as the result of a lesion of the association tracts between both occipital lobes and the left first temporal convolution at a point where these tracts are near together in the white matter of the left occipital lobe. This explanation is by no means generally accepted. The symptom-complex is not always typical, but Bruns says it has been fairly typical in a number of cases, and was so in one that he himself observed. Mind-blindness is present to some extent in association with the optic aphasia, but lesions of both occipital lobes seem to be requisite for this sign to be pronounced. The reader is referred to the chapter by Dr. C. K. Mills on the " Psychology of the Visual Act and the Focal Diseases of the Visual Cortex" for a fuller discussion of achromatopsia, mind-blindness, optic aphasia, word-blindness, alexia, and allied subjects.

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[4] De Schweinitz, New York Med. Journ., May 2, 1891, p. 514.

[5] Bregman, Deutsche Zeitschrift für Nervenheilkunde, vol. xxvi., Nos. 4 to 6, p. 525.

Transitory amaurosis sometimes occurs with tumors of the occipital lobe (Bruns), but according to Hirschberg it has little localizing value, as has also conjugate deviation of the eyes, which according to Oppenheim occurs sometimes with occipital lobe tumors. There may be a centre for associated ocular movements in the parieto-occipital lobe as well as in the frontal lobe.

Hemianopsia.—The most valuable localizing sign of tumor of the occipital lobe is homonymous lateral hemianopsia. This by itself is not sufficient, inasmuch as it may be caused by a lesion of the visual pathways anywhere between the chiasm and the visual cortex of the occipital lobe. According to Oppenheim, Bernhardt in fifteen cases of tumor of the occipital lobe found hemianopsia in only two cases. An examination of the more recent reports has shown that rarely hemianopsia is absent in cases of tumor of the occipital lobe.

It has been supposed by some investigators that if the hemianopsia is caused by a lesion of the visual cortex the patient will be ignorant of his half-blindness until it is pointed out (" vision nulle" of Dufour), whereas this will not be the case if the hemianopsia is caused by a subcortical lesion. This distinction is unreliable. Harris has examined for this sign several times without any satisfactory results, and in one case in which the lesion was in the posterior part of the internal capsule, the patient was not aware of any defect of vision, and could with difficulty be persuaded that considerable portions of his visual fields were lost. (See Chapter IV, p. 167.)

It seems strange that hemianopsia could be overlooked, and yet a case has been mentioned to me by Frankl-Hochwart, in which the sign was discovered after it was noticed that the patient, then in a large hospital, habitually ate only the food on one half of his plate. It was found on examination that he saw only one half of his plate.

Hemiachromatopsia.—Hemiachromatopsia has been considered by some as the result of lesion of certain special centres or fibres in the visual system. Ward A. Holden refers to a considerable number of cases in which, with a normal field for large white test-objects, there was hemianopsia for colors. Most of the older writers agree that there must be special separate cortical centres for the perception of form, light, and color, and that in the cases under consideration, the cortical color centre was exclusively involved. This supposition was defended particularly by Samelsohn, Landolt, and Wilbrand. Of late there has been a tendency to depart from this teaching, and Ole Bull and Otto Dahms do not accept it. Some observations of his own have convinced Holden that the supposition of there being a lesion of some special color centre in these cases is wrong. His views are as follows:

"A slight interference in conduction of any of the fibres of the visual tract, leads to an inability to recognize green or even red, or to distinguish slight differences in luminous intensity. A more marked disturbance in conduction leads to the inability to recognize blue, or to distinguish quite marked differences in luminous intensity. A greater interference with conduction prevents the distinguishing of white from black, and with complete interference with conduction even perception of light is lost.

"Thus the recognition of color varies with the light sense, and the assumption of the involvement of a particular cortical color centre in cases of hemiachromatopsia is not only unnecessary, but is palpably erroneous."[6]

De Schweinitz[7] expresses the opinion that homonymous hemiachromatopsia probably is caused by a cortical lesion of less intensity than one which produces absolute hemianopsia. In a case studied by him at the onset the hemianopsia was absolute: later light sense and form sense returned. The obliteration of the color sense remained, although in all other respects the patient had recovered. Non-cortical lesions, he holds, may also produce hemiachromatopsia.

An interesting case recorded by Harris shows that a lesion in the neighborhood of the internal capsule may cause hemiachromatopsia, and that the lesion need not be cortical to produce this sign. In his case there were almost complete left hemiplegia, partial left hemianæsthesia, and complete loss of all color perception, even of large area, in the left lower quadrant for each eye; small objects, as the head of a pin or a disk of white paper 1 centimetre in diameter, were also invisible, and the patient could not distinguish between one and two fingers held up in the same area, though he could readily do so in each of the remaining quadrants. The cuneus was found to be normal on each side, but there was an area of softening in the right internal capsule.

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[6] Holden, Archives of Ophthalmology, vol. xxiv. p. 447.

[7] Diseases of the Eye, p. 559.

Harris quotes from Swanzy's " Handbook of the Diseases of the Eye and their Treatment:" " It is now generally believed that relative hemianopsia (*e.g.*, color hemianopsia alone) is the result of a lesion of less intensity than that which causes absolute hemianopsia . . . and that the color sense is more easily affected by disease than the form or light sense, and that, too, irrespective of the position of the lesion in the visual path."

Hemianopic Pupillary Reaction Sign.—The absence of the hemianopic pupillary rigidity of Wernicke points rather to a tumor of the occipital lobe when a lesion of the visual pathways is probable, as shown by lateral homonymous hemianopsia, but the sign is difficult to obtain and the danger of error is great. I believe, however, that I have seen this sign positively demonstrated in a few cases. It consists in contraction of the iris to light only when the halves of the retinæ still preserving visual function are illuminated. As there is no light reflex from illumination of the blind half of each retina when this sign is present, the lesion must be somewhere within the pupillary reflex arc, therefore in or below the primary visual centres (external geniculate body, pulvinar of the optic thalamus, and anterior colliculi of the corpora quadrigemina). This sign cannot be caused by a lesion above the primary visual centres, in the optic radiations or visual cortex, although there may be hemianopsia from such a lesion. (See Chapter IV, p. 170, and Chapter VI, p. 318.)

Paralysis of Cranial Nerves and Cerebellar Ataxia as Signs of Occipital Tumor.—When paralysis of cranial nerves occurs in association with homonymous lateral hemianopsia it is in favor of a growth below the occipital lobe, and yet it is not absolute proof of a basal lesion, especially when the paralysis implicates the sixth nerve of either side. This nerve has so long a course within the cranium that it may be injured by increased intracranial pressure. Oppenheim believes it is possible for the nerves of the posterior cranial fossa to be affected by the pressure of an occipital tumor.

Jacksonian motor epilepsy cannot be regarded as a common sign of tumor of the occipital lobe. Cerebellar ataxia has been observed, and has been explained as the result of pressure upon the cerebellum.[8] The tentorium usually is sufficient to prevent pressure upon the cerebellum, but in a case of internal hydrocephalus in the service of Dr. Mills, caused by closure of the aqueduct of Sylvius, which I had the opportunity to study and have reported, pressure on the cerebellum from the distended posterior horns of the lateral ventricles seemed to afford a satisfactory explanation of the ataxia. This case is as follows: A boy, aged nineteen years at the time of death, had had some headache once or twice a week, with vomiting, since childhood; but about six months before death the headache became severe, and cerebellar gait was observed, and he staggered like a drunken man. The station with feet apart was good, but with feet together was poor, and the sway was not increased by closure of the eyelids. The gait was so unsteady that at times he had to catch hold of the bed to keep from falling. He was occasionally dizzy when lying on his back, and the dizziness was relieved by turning on his side. Slight exophthalmos was observed. Distinct optic neuritis was present in each eye. The patellar reflexes were exaggerated. The Babinski reflex was absent on each side. All venereal disease was denied. There was certainly sufficient here to suggest a cerebellar lesion, but none existed.

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[8] L. Bruns, Die Geschwülste des Nervensystems. S. Karger, Berlin, 1897.

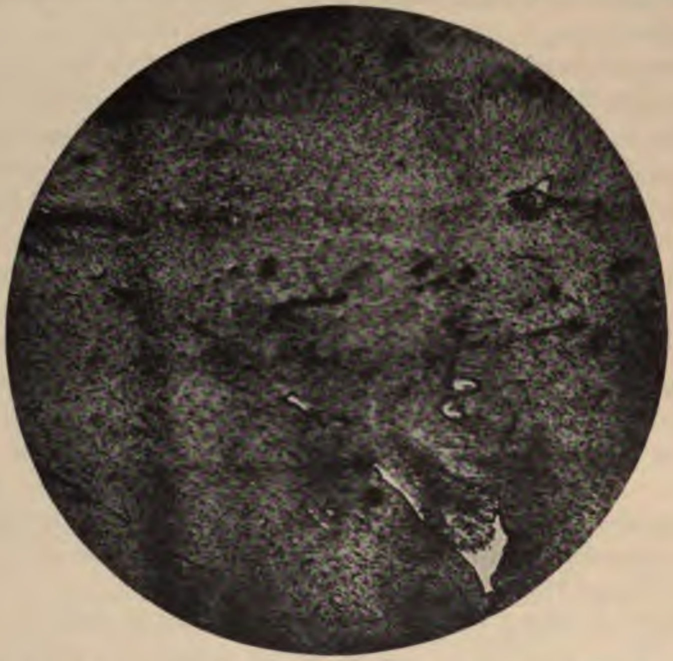

FIG. 1.—Almost complete closure of the aqueduct of Sylvius, only the extreme lower end remaining patulous, causing internal hydrocephalus, and symptoms of cerebellar tumor. The fourth ventricle was normal in size.

On removing the skullcap, which was extremely thin, the brain and its membranes bulged outward. On the internal surface of the skullcap the moulding of the convolutions of the brain could be distinctly made out. The third and lateral ventricles were distended with clear fluid, and the aqueduct of Sylvius was closed by proliferated neuroglia, while the fourth ventricle was of normal size. In microscopical sections a very small opening where the aqueduct should be, was found, but during life this may have been closed, and it is doubtful whether it extended all the way from the third to the fourth ventricle.

In a case of echinococcus cysts of the left occipital lobe reported by Mingazzini, lateral homonymous hemianopsia was not mentioned by the patient and was not searched for by the physicians. Choked disks, much diminished patellar reflexes, intense headache occurring periodically, and tinnitus in the left ear were rather suggestive of the cerebellum as the seat of the tumor, and Mingazzini says that if lateral homonymous hemianopsia had existed one might still think of cerebellar tumor, as this phenomenon as a distant symptom has been observed in tumor of the cerebellum.

Lumbar puncture was made in this case and the patient went into collapse the same evening and died the next morning. This procedure is dangerous in cases of tumor of the occipital lobe, cerebellum or medulla oblongata, and probably also when the tumor is situated elsewhere in the brain.

A similar case has been observed by Mann. A tumor of the occipital lobe was found. Hemiataxia and the Babinski sign occurred on the same side as the tumor, and there was much in this case to suggest a tumor of the cerebellum.

Oppenheim speaks of a case of Schultze in which a cerebellar tumor by pressure on the occipital lobe caused symptoms of a lesion in the occipital lobe. He refers to a case of his own in which a tumor of the precuneus caused cerebellar symptoms by pressure. A tumor of the cerebellum was observed by him to compress the descending horn of the lateral ventricle, and thereby to cause distention of this horn and interference with the function of the occipital lobe. I have seen distention of one posterior horn of the lateral ventricle caused by a tumor within the pons.

TUMORS OF THE PARIETAL LOBE.

Tumors in this area of the brain may cause convulsions in the limbs and face of the opposite side by irritation of the adjoining motor area, weakness of the limbs of the opposite side from pressure upon the cortical motor centres, loss of stereognostic perception and of the sense of position, and loss or impairment of tactile sensation with less implication of pain sensation, and awkwardness of movements in the limbs of the other side. The astereognosis and loss of the sense of position may be so great that the patient, as in a case under my care, may be unable to recognize any object placed in his hand of the opposite side when the eyes are closed, or have any knowledge of the position of the limbs of the opposite side, and may take the hand of the examiner believing it to be his own. In the case referred to the meningitis in plaques was found over and almost confined to the opposite parietal lobe. Ataxia of the limbs on the side opposite the lesion is to be expected if the sense of position is so impaired that the patient has no knowledge of the position of his limbs. I have, in several cases of lesion of the parietal lobe, observed the patient place the normal hand on his shoulder or some part of the opposite upper limb in searching for the hand of this side, and by following the limb down reach his hand.

Although I believe that a lesion of the parietal lobe may cause ataxia, loss of stereognostic perception, and loss of the sense of position and some disturbance of tactile and possibly of pain sensation in the limbs of the opposite side, I have not been convinced that a lesion of the motor area, or at least of the post-central convolution, will give none of these symptoms. The tendency now is to regard the post-central convolution as having no motor function.

Marinesco remarks that Jonesco removed a part of the cortical centre for the left upper limb in a man. After the operation the patient could move his shoulder and elbow very well, but not his hand. Nine months later the movements of the left hand were very imperfect, and tactile sensation, the sense of position, and stereognostic perception were diminished in the left hand. In another case Jonesco removed the centres of the face and upper and lower limbs of the right cerebral cortex. A year after the operation there was much weakness of the left side of the body, and the sense of position, stereognostic perception, and tactile sensation were affected in the left upper limb. Thermal and painful sensations were preserved. These operations were done for epilepsy, and in one case, at least, and probably in both, the cortical motor areas were determined by the electric current.

I have observed loss of stereognostic perception caused by lesions much below the cortex of the brain,—viz., in a case of tumor of the pons (Potts and Spiller) and in a case of tumor compressing the medulla oblongata (case of Dercum); but where the symptoms indicate a tumor of the cerebrum that probably is cortical, I believe the loss of stereognostic perception and of the sense of position and ataxia of the upper limb of the side opposite to that in which is the lesion indicate much implication of the parietal lobe.

In 1898, at a meeting of the Philadelphia Neurological Society, I[9] referred to a case that I had seen in the service of Dr. Lloyd, at the Philadelphia Hospital, which seemed to show that the cortical representation of the sense of position is in the parietal lobe. A man complained, during the night preceding his attack, of fatigue, headache, and inability to sleep. The following morning he fell to the floor on attempting to rise from his bed, but was not unconscious. When he was found he had paresis of the left limbs and of the left side of the face and tongue. Later the patient could raise his left upper limb above his head, but every movement of this limb was ataxic in an extreme degree, though the limb was not paralyzed. The mental condition of the patient prevented an examination of the condition of sensation. At the necropsy a hemorrhage was found in the right parietal lobe. The hemorrhagic area was about 2 centimetres in diameter, and extended inward in the form of a cone to the lateral ventricle, having its base in the cortex. It was situated about 4 centimetres from the longitudinal fissure and about 2 or 3 centimetres behind the Rolandic fissure. The brain had been cut into frontal sections about 1 centimetre apart, when I had an opportunity to examine it, and, although the injury to the cerebral tissue prevented an exact determination of the location of the hemorrhage, it was evidently in or very close to the supramarginal gyrus. The ataxia was probably caused by a loss of the sense of position.

This was probably one of the first cases reported in this country, showing that ataxia from a loss of the sense of position may be caused by a lesion of the parietal lobe. The case of Starr and McCosh preceded it.

A case that Dr. C. K. Mills and I saw together, June 3, 1901, in consultation, the case being under the charge of Dr. Ida Richardson, affords evidence also of the localization of the stereognostic perception and of the sense of position in the parietal lobe. The patient's left upper limb was decidedly ataxic, paretic, and astereognostic, and the head was retracted and rigid. The necropsy was made by Dr. W. F. Hendrickson. An area of depression, indefinite in outline, but about 6 centimetres in diameter, was found in the parietal lobe of the right cerebral hemisphere just posterior to the fissure of Rolando. Palpation revealed less resistance at this point than over the surrounding tissue. On section this area was found to be the seat of extensive softening. The process implicated practically the entire right parietal lobe.

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[9] Journal of Nervous and Mental Disease, January, 1899, p. 43.

The case reported by Dr. C. K. Mills,[10] which was seen by me and others in consultation, was one of the first to establish the localization of the stereognostic perception in the parietal lobe. A diagnosis of tumor of this lobe was made because of the loss of the sense of position, impaired cutaneous sensibility, astereognosis, and ataxia. The case has been so fully reported by Dr. Mills and is so well known that I need not refer to it further.

Oppenheim,[11] after giving a description of his case of parietal tumor with operation, remarks that lesions of the parietal lobe cause ataxia of one or both limbs of one side. Irritative or paralytic motor symptoms are absent, or when they occur they are the result of pressure. The sensory disturbance is always a partial hemianæsthesia, in that pain and temperature sensations are preserved, while tactile sensation and especially the sense of position and the stereognostic perception are impaired or lost. Such a symptom-complex occurs when the lesion does not extend far into the white matter of the parietal lobe. He says, also, that the cases in which tumor of the parietal lobe has been so accurately diagnosed that a radical operation was possible are very few.

This case of Oppenheim was reported by him February 12, 1900, and published in the third number of the *Mitteilungen*.

In his monograph [12] on tumors of the brain he says that in the majority of cases of tumor of the parietal lobe sensory, irritative and paralytic phenomena were observed. He refers to Mills's case, but adds that the cases are not sufficient to permit us to regard the above-mentioned phenomena as positive evidence of lesions of the parietal lobe. The evidences of the localization value of these symptoms are, however, rapidly accumulating.

In March, 1901, Dr. C. W. Burr [13] reported a case in which there were slight hemiplegia, slight tactile anæsthesia, astereognosis, mind-blindness, loss of the temperature sense on one side, sensory aphasia varying in intensity, and mental dullness. In this case a tumor was found in the parietal lobe, pressing against but not invading the ascending parietal convolution, partially destroying the angular gyrus, and involving almost the entire posterior parietal lobule.

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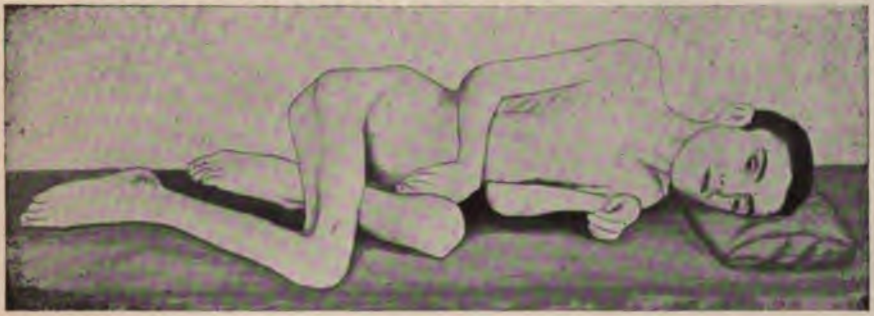
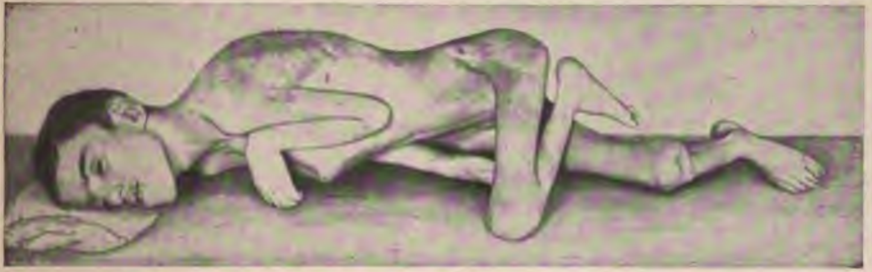
[10] Mills, Keen, and Spiller, Journal of Nervous and Mental Disease, May, 1900, p. 244.

[11] Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie, 1900, vol. vi. pp. 382, 383.

[12] Oppenheim, Die Geschwülste des Gehirns, second edition, p. 116.

[13] The Amer. Journ. of the Med. Sci., March, 1901, p. 306.





FIGS. 2, 3.—Extreme muscular atrophy, hemiplegia, and bilateral contractures from internal hydrocephalus confined to one side of brain.

A tumor of the parietal lobe by growing downward may involve the internal capsule and the optic radiations, and thereby cause hemiplegia, hemianæsthesia, and hemianopsia.

TUMORS OF THE TEMPORAL LOBE.

When the tumor is situated in the left temporal lobe, sensory aphasia from implication of the posterior part of the first temporal convolution may be expected. The patient becomes word-deaf and is unable to speak connectedly; he may also be word-blind. The latter symptom, according to some neurologists, is to be explained by implication of the left angular gyrus. The right temporal lobe is a much more "silent area" than the left, at least in right-handed people, and a tumor here may grow to a considerable size before causing localizing symptoms. A tumor in either the right or left temporal lobe may by its growth cause pressure upon, or direct involvement of, the posterior limb of the inner capsule, with hemiplegia of the opposite side of the body.

TUMORS OF THE MOTOR AREA.

A growth in this region is more easily diagnosticated than one in any other part of the brain. Jacksonian motor epilepsy with paresis or paralysis of the face and limbs, and with motor aphasia, if the tumor is on the left side in a right-handed person, forms a symptom-group very characteristic of tumor in the central gyri. I have observed a case in which a gradually developing hemiplegia, with paresis of the other lower limb, resulted from internal hydrocephalus confined to one lateral ventricle. Such a case might be difficult to diagnose from one of subcortical tumor of the motor region (Figs. 2, 3).

TUMORS OF THE FRONTAL LOBE.

The frontal lobe may be seriously involved by a tumor without causing localizing symptoms, but it is probable that a growth is more likely to cause derangement of the higher intellectual functions when it is situated in this portion of the brain, especially in the left cerebral hemisphere. Judgment, power of concentration of thought, ability to understand what is read, etc., may show early impairment in cases of frontal tumor.

Jacksonian convulsions in the limbs of the opposite side are not uncommon, because of the proximity of the motor area, and hemiplegia or monoplegia of the opposite side may also be produced by pressure upon, or direct implication of, the motor region of the brain. According to the investigations of Grünbaum and Sherrington, the centres for the limbs and face are entirely in front of the central fissure.

TUMORS OF THE OPTIC THALAMUS.

According to some neurologists, we may expect as signs of a lesion of the optic thalamus a hemiplegia which in some cases may be slight, in others intense, but even then is likely to diminish in intensity, and often is less pronounced than is the hemianæsthesia on the same side as the paralysis. These symptoms are explained by the theory that all sensory fibres pass through the optic thalamus, and therefore a tumor growing in this ganglion directly implicates the sensory tract, and causes a disturbance of motion, at first by pressure upon the internal capsule, and later by direct implication of this system of fibres. The case that I had the opportunity of studying with Dr. Dercum some years ago, in which objective disturbance of sensation had existed many years, and a cyst was found in the *carrefour sensitif* of Charcot and in the lenticular nucleus, without any implication of the optic thalamus, has led me to believe that the sensory fibres probably do not all terminate in the optic thalamus. In addition to the disturbances of motion and sensation mentioned, subjective disturbances of sensation,—viz., pain, also choreiform or athetoid movements, forced laughter, hemianopsia, disturbances in the emotional innervation of the opposite side of the face in laughing and crying, and forced movements,—may be expected.

In a case of abscess of the right optic thalamus, C. E. Beevor[14] observed severe pain in the occipital region of the same side, vomiting, double optic neuritis, with some weakness of the face and limbs of the opposite side, a tendency of the patient to [illegible] to the side of the lesion, in the direction of [illegible] and a tendency to fall backward and to the right.

He regards as symptoms of [illegible] thalamus occipital pain on the same side [illegible] weakness and nystagmus [illegible] weakness of the opposite limbs [illegible] staggering backward [illegible] with the [illegible]

[illegible] thalamus [illegible]

[illegible] of certain fibres [illegible] of the [illegible] hemichorea to a lesion of the optic thalamus; Kahler and Pick to irritation of the pyramidal tract, but Bonhoeffer [15] believes that hemichorea depends upon a lesion of the superior cerebellar peduncle, and he attempts to show that in most of the cases with necropsy in which hemichorea was present these peduncles were implicated.

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[14] [illegible]

The diagnosis of tumor of the optic thalamus not infrequently is one of great difficulty and in some cases it is impossible. The symptoms may be those of a cerebellar tumor as in a case observed by Douglas Stanley,[16] in which a large glio-sarcoma was found occupying the whole of the left optic thalamus, causing compression of the internal capsule and displacement outward of the lenticular nucleus, and projecting into the lateral ventricle, so as to cause slight pressure on the opposite optic thalamus. The patient was a little weak in the right arm and leg, and had difficulty in controlling volitional movements of these limbs. He had a coarse, irregular tremor of the right arm. His gait was unsteady and he reeled to the right side. On attempting to stand with his feet together he was liable to fall to the right side. Occasionally convergent strabismus appeared on the right side, and occasionally he had internal squint with the left eye varying in degree, so that there was alternating converging strabismus, and it was often difficult to say which external rectus was most affected. This case shows that paralysis of an external rectus may be a distant symptom, and I have seen it in a case of tumor of the parietal lobe.

The occurrence of Jacksonian epilepsy from a tumor situated deeply in the brain may cause a mistake in diagnosis, but in rare instances convulsions of this character may occur, as in a case reported by J. M. Clarke, in which a tumor was confined to the optic thalamus. Paralysis nowhere was absolute, and in the face was slightly marked, and there was no deviation of the tongue. There was rigidity but no ankle clonus, nor exaggeration of reflexes. Loss of control over bladder and rectum existed. In these respects the group of symptoms, he says, corresponded to those observed by Bastian. In addition there was left ptosis, dilated pupils, with loss of reaction to light, turning of the head first to the left and afterward to the right side, with mobile spasm of the right arm and leg, and vasomotor phenomena of the right arm.

In Clarke's case the head looked towards the side of the lesion in the earlier stages and only subsequently was directed towards the healthy side.

According to the experiments of Schiff and Nothnagel in unilateral lesions of the thalamus the head looks towards the healthy thalamus, while on the side of the healthy thalamus the flexors of the upper extremity, and on the side of the lesion the extensors of the upper extremity appear contracted.

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[15] Bonhoeffer, Monatsschrift für Psychiatrie und Neurologie, vol. i. p. 6.

[16] Stanley, Brain, 1898, vol. xxi.

In Clarke's case the positions of the right arm and leg exactly corresponded to those commonly observed in ordinary cases of hemiplegia from hemorrhage into the internal capsule, and he believes they were accounted for by pressure of the tumor on the inner capsule. During the short time the patient lived after an operation the symptoms of paralysis and spasm disappeared in the face and arm, and almost entirely in the leg. Any effect obtained by operation was believed to be by relief of pressure, and would seem to indicate that pressure on the internal capsule was the essential agent in the production of paralysis and spasm.

Clarke[17] describes in this case convulsive seizures, beginning in the right arm and affecting the right arm and leg, lasting for two or three minutes and followed by rigidity of these limbs for the same length of time, or of spasm confined to the right arm occurring at irregular intervals. These attacks seem to have been of the Jacksonian type, although the lesion was in the optic thalamus. Later the spasmodic movements of the right upper limb were continuous. He describes also an intention tremor among the early symptoms.

Usually Jacksonian motor epilepsy indicates a lesion in or near the motor cortex, but I have found this sign in a case of my own, caused by a tumor deep within the white matter of the brain but above the internal capsule.

A tumor of the optic thalamus does not always cause disturbances of vision, although it would be expected to do so because of the probable situation of visual fibres within the pulvinar, and because of the liability to interference with the flow of cerebrospinal fluid through the aqueduct of Sylvius. In a case observed by Byrom Bramwell,[18] a diagnosis of cortical gumma in the position of the right lower limb centre was made on account of tenderness over the leg and arm centres on the right side; and of attacks of Jacksonian epilepsy occurring several times a day, the spasm beginning in the left big toe; and because of exaggeration of the left knee-jerk. Other symptoms of tumor were present. An examination at the necropsy revealed a gliosarcoma about the size of a small egg in the right optic thalamus and encroaching slightly upon the motor fibres of the pyramidal tract. The tumor was thought to have produced irritation of the leg fibres of the internal capsule, but it seems very strange that a tumor so deep within the brain as in this and in Clarke's case should have caused Jacksonian epilepsy. Bramwell says that in four cases in which the optic thalamus was invaded by a new growth he was unable to detect any definite symptoms due to a lesion of this area, unless paroxysms of flushing, which were very marked in one case, were due to this cause.

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[17] J. M. Clarke, Lancet, 1891, p. 1283.

[18] Byrom Bramwell, Brain, 1899, vol. xxii. p. 49.

In a case of tumor of the posterior portion of the left optic thalamus and pressing on the inner capsule, reported by Major,[19] there was no optic neuritis and no color-blindness. The symptoms were paralysis of the right limbs and right side of the face, numbness of the right side, dilatation of the right pupil and right homonymous hemianopia.

TUMORS OF THE CORPORA QUADRIGEMINA AND CEREBRAL PEDUNCLES.

If we recall the structure of the region of the corpora quadrigemina we can understand that ocular palsies are usually among the early manifestations of tumor of this part of the brain. The nuclei of the oculomotor and trochlear nerves lie near the aqueduct of Sylvius, and therefore pressure upon these nuclei, or direct implication of them by a tumor causes partial paralysis of the muscles of the eyeball on each side. Ataxia may be attributed to the involvement of the superior cerebellar

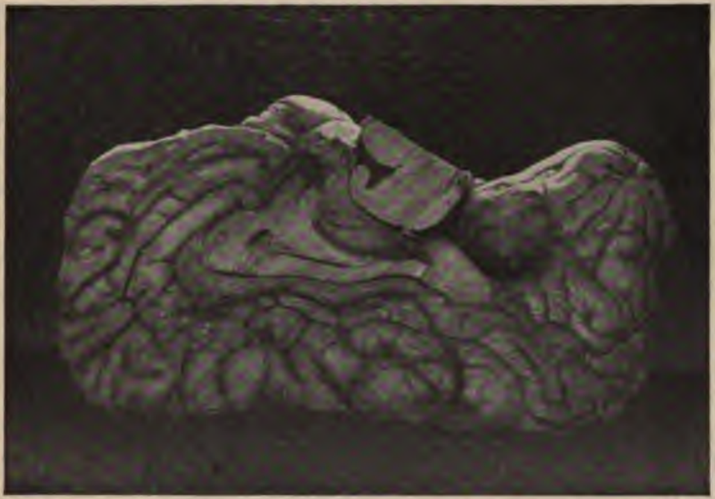

Fig. 4.—Fibroma on the corpora quadrigemina. The right cerebral hemisphere, and the posterior part of the pons have been removed.

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[19] Brit. Med. Journ., April 16, 1892, p. 818.

peduncles which decussate below the aqueduct and form the red nuclei. Disturbance of hearing may also be expected, and may be explained by the implication of the posterior colliculi of the corpora quadrigemina. The inner muscles of the eye, the iris and ciliary muscles, may escape, because the position of the nuclei for these muscles is probably in advance of those for the external muscles of the eyeball, in fact, rigidity of the iris is not a common sign of tumor in this part of the brain, and ptosis from involvement of the levator palpebræ also may be absent. Inequality of the pupils has been observed from tumor of the corpora quadrigemina. Occasionally absence of convergence-reaction and paralysis of accommodation have been seen. Oppenheim reports recurrent ophthalmoplegia.

In Kolisch's case of tubercle of the corpora quadrigemina and pons both oculomotor nerves were paretic, but the pupillary reaction was preserved. The tumor did not extend forward beyond the corpora quadrigemina, and probably for this reason the pupillary reaction was preserved.

In a case of tuberculous tumor confined to the corpora quadrigemina reported by Bristowe, the tumor lay between the upper surface of these bodies and the aqueduct of Sylvius. All the movements of the eyes were more or less imperfect, except possibly downward movement. The irides reacted well to light, but reaction to accommodation could not be determined. Mr. Nettleship, who made the ophthalmological examination, thought that the centres for the iris and ciliary muscles had escaped: This case also seems to show that these centres are in advance of the other oculomotor centres.

In B. Sachs's case of tubercle of the corpora quadrigemina there was almost complete bilateral external ophthalmoplegia, but during the entire period of observation the ciliary muscles and irides remained exempt. Sachs regards this as striking proof that the nuclei of these muscles are at some distance from those governing the external ocular muscles.

The internal ophthalmoplegia in Gordinier's case from a lesion far back in the oculomotor nuclei is remarkable, as the nuclei for the internal ocular muscles are usually placed well forward, by L. Bruns, in the median side of the optic thalami.

In a case of tumor which seems to have been confined to the aqueduct of Sylvius, and was reported by J. Collins,[20] the gait was like that of an intoxicated person, but there was no ocular palsy and hearing was

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[20] Collins, Amer. Jour. of the Med. Sci., 1895, vol. cx. p. 420.

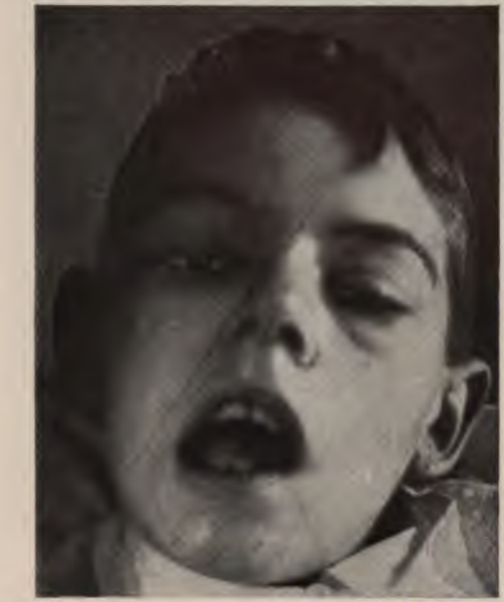

FIG. 5.—Glioma of the right side of the pons. Attempt to converge. Left eye is moved more toward the right than when the attempt is made to move both eyeballs to the right (Fig. 7). Marked deviation of lower jaw to the right on account of paralysis of the right pterygoid muscles. Associated ocular palsies.

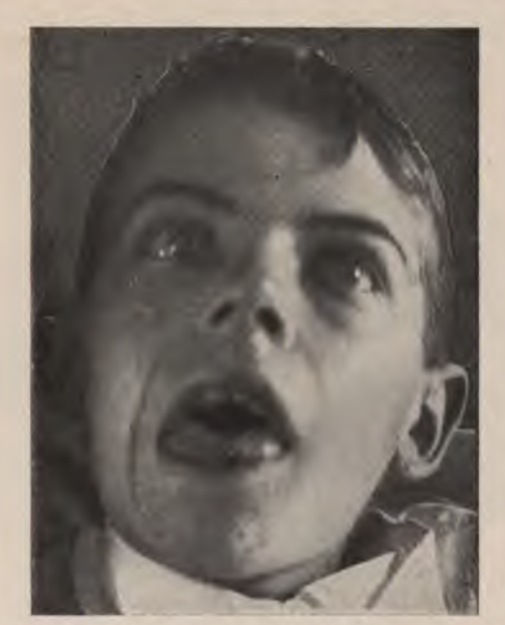

FIG. 6.—Glioma of right side of the pons. Attempt to look upward and to protrude the tongue. The eyeballs move very slightly upward. The lower jaw goes to the right, the tongue to the left when protruded. Figs. 5, 7, 8 and 18 are from the same case.

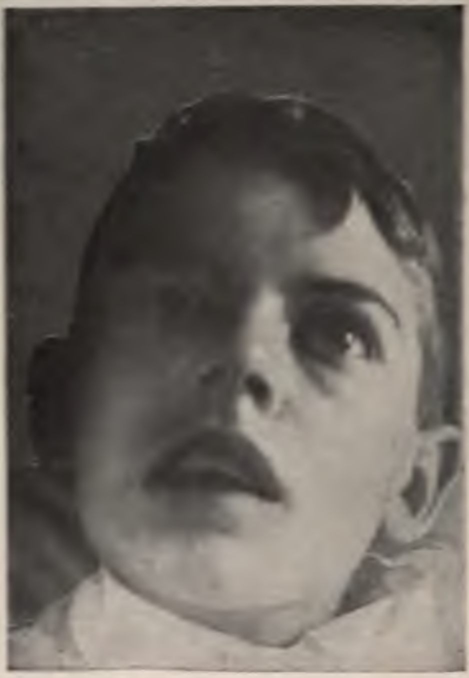

FIG. 7.—Glioma of the right side of the pons. Attempt to look to the right and to separate the lips with the teeth together; weakness in the lower distribution of the seventh nerve on each side.

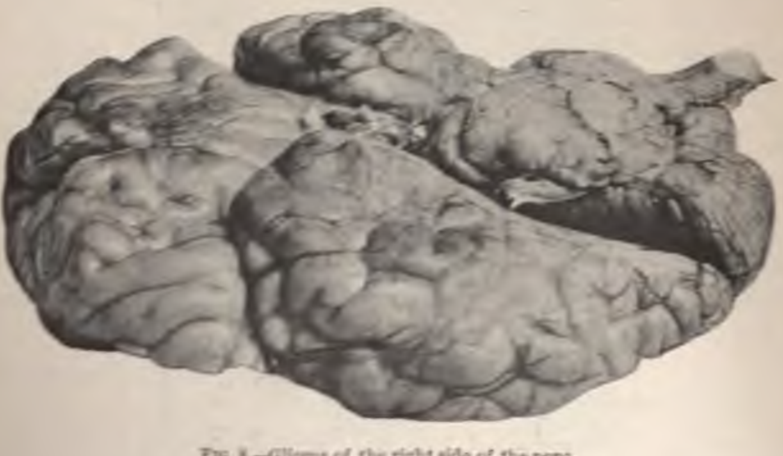

FIG. 8.—Glioma of the right side of the pons.

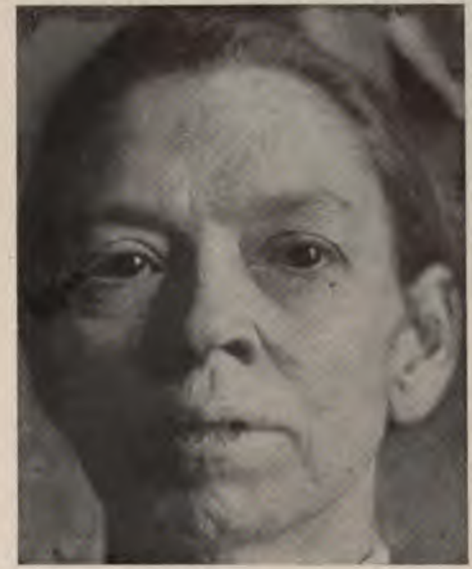

FIG. 9.—Case seen with Dr. J. W. McConnell. Lesion probably near the aqueduct of Sylvius; attempt to converge; power of convergence lost.

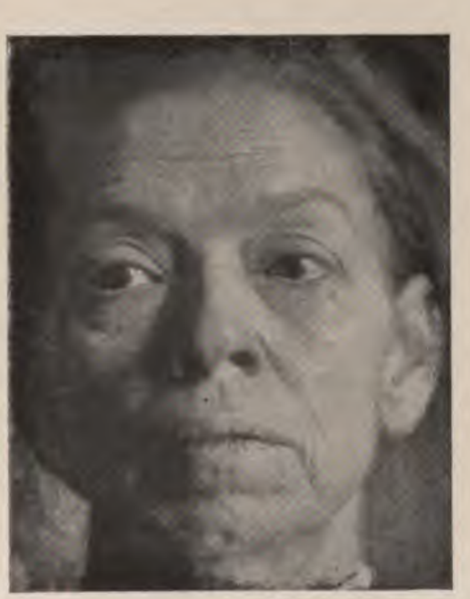

FIG. 10.—Same case as shown in Fig. 9. Attempt to look upward shows failure of eyes to move upward; wrinkling of forehead in the attempt is well shown; paralysis of upward associated movements.

good. We may assume that pressure from the tumor was not sufficient to cause many symptoms. The third and lateral ventricles were much distended, but the fourth ventricle contained no fluid and presumably was not dilated. The case resembles one to which I have referred in my section on tumors of the occipital lobe, of occlusion of the aqueduct of Sylvius by proliferation of the neuroglia.

Associated Ocular Palsies.[21] — Disturbances in the associated movements of the eyes (Blicklähmung) afford a sign of localizing value in lesions of the brain, and yet this subject has received comparatively little attention. Von Kornilow, who has written the best recent paper on these palsies, remarks that clinical cases carefully studied are rare, and even more so are those with necropsy. (See Chapter V, p. 266.)

The opinion of Parinaud and Sauvineau that upward or downward associated ocular paralysis is always associated with paralysis of convergence is incorrect, as shown by a case reported by Parinaud himself, by a case reported by von Kornilow, and by other cases. It is true, however, as Parinaud pointed out in 1883, that in paralysis of associated ocular movements diplopia often is absent. Indeed when in such palsy diplopia later appears it may be a sign of improvement, as it was in a case observed by me, where the disappearance of the palsy in one eye caused diplopia, soon followed by complete recovery. Although paralysis of associated ocular movement of any form is usually a sign of grave significance, one should be careful about giving a fatal prognosis. In a case reported by Raymond and Cestan [22] the paralysis lasted ten years, and in a number of cases complete recovery has occurred, while in others improvement has been very marked, as in some of the cases studied by me.

The evidence is strong that paralysis of associated lateral movements of the eyeballs is indicative of a lesion of the posterior longitudinal bundle near the sixth nucleus. The external rectus muscle on the side of the lesion may be more affected than the internal rectus of the other eye, and this according to Parinaud [23] is the rule. He mentions also that the movement of convergence may be greater in the eye in which the external rectus is paralyzed, because of the paralysis of the external rectus.

The disturbances of the upward or downward associated movements have been studied less than those of lateral associated movement. The former, though usually acquired, may be congenital, and Oppenheim[24] has observed congenital paralysis of associated upward movement. Uhthoff[25] speaks of paresis of upward associated movement with slight impairment of movement in other directions, occurring in multiple sclerosis.

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[21] Adapted from the author's Presidential Address, read at the meeting of the American Neurological Association, June, 1905, and published in the Journal of Nervous and Mental Disease, July and August, 1905.

[22] Revue Neurologique, 1901, p. 70.

[23] Archives de Neurologie, vol. v. No. 14, 1883, p. 145.

We have reason to believe that the cerebral cortex exerts some control over the associated movements of the eyeballs; especially strong is the evidence concerning lateral conjugate movements. Conjugate deviation of the eyes has been studied within the past few years by Klaas,[26] and from his investigations of the literature, it appears that this phenomenon was first observed by Andral (1834). It was studied by Duplay, Durand-Fardel, and others, but Prevost showed that conjugate deviation of the eyes often occurs in hemiplegia and has nothing to do with strabismus, as Andral believed; he showed further that it is often associated with deviation of the head; that the deviation is toward the lesion when the lesion is cerebral, and away from the lesion when it is in the isthmus. Landouzy believed that irritation and paralysis produce deviation in opposite directions when the lesion is in the cerebrum, and Grasset showed that the patient looks at his paralyzed limb when the lesion is a paralyzing one in the brain stem, and at the lesion when it is an irritating one, and in this part of the brain. Wernicke assumed that the rapid disappearance of conjugate deviation of the head and eyes is owing to the partial control of the lateral ocular movements by the cerebral hemisphere of the same side, and to the action of this hemisphere in place of the injured opposite cerebral hemisphere. Wernicke believed that conjugate deviation of the eyes is caused by a lesion of the lower parietal lobe, and he reported three cases of lesion in the lower part of each parietal lobe in which voluntary lateral associated ocular movements were lost, pseudo-ophthalmoplegia, although in another case with similar lesions disturbance of the ocular movements was not observed.

The evidence concerning a centre for lateral movements of the eyes in the lower parietal region or angular gyrus is conflicting. There is more evidence that a centre for lateral associated ocular movements is situated in the posterior part of the frontal lobe.

The recent investigations of Grünbaum and Sherrington by electrical irritation have again demonstrated that a centre for ocular movements probably exists in the frontal lobe. Pathological alteration of the frontal lobe has given evidence to the same effect. In a case of cerebral syphilis with severe lesion of the right frontal lobe, reported by Karl Schaffer,[27] paralysis of associated movement of the eyeballs to the left occurred, and Schaffer concludes from a study of this case that a centre for lateral associated movements exists in the posterior part of the second frontal convolution, although he does not deny that there may be another and similar centre in the supramarginal and angular gyri.

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[24] Lehrbuch der Nervenkrankheiten, 4th edition, vol. ii. p. 706.

[25] Archiv. für Psychiatrie, vol. xxi. 1890, p. 379.

[26] Uber konjugierte Augenablenkung bie Gehirnkrankheiten, Inaugural Dissertation, Marburg, 1898.

Hemianopsia cannot be regarded as the cause of conjugate deviation in all cases, although much has been attributed to it recently by certain French writers. Dejerine and Roussy[28] have demonstrated that it is not necessarily the cause of the deviation, inasmuch as deviation existed in a patient of theirs who had been born blind and whose cortical visual zone had not been developed.

So far the knowledge gained relates chiefly to associated lateral movements, but we know much less concerning a cortical centre for upward or downward associated movements.

The views of Parinaud and some other writers on this subject lack confirmation. They believed that there is a centre for ocular movements in the occipital lobe and a more important one in the frontal lobe. The inferior part of the latter centre controls the upward movement of the eyes, the superior part controls the downward movement of the eyes, the intervening part controls the lateral movements. It is true that lateral associated movements have been impaired with paralysis of upward or downward associated movements in a number of instances, as they were in some of my cases, but a lesion in the vicinity of the corpora quadrigemina will better explain this form of paralysis than a lesion of the cerebral cortex. Teillais says, regarding his own case, it would be unreasonable to regard the lesion as cortical, because if the lower part of the cortical centre controls the upward movements, and the upper part the downward movements, then the whole of this centre except the middle portion must have been destroyed, as the lateral movements were preserved. Such a view cannot be accepted.

Paralysis of lateral associated movement from cortical lesion, as seen in the conjugate deviation of the eyes, is always, so far as I know, transitory, and the persistence of such paralysis points to a lesion of the posterior longitudinal bundle. We have little anatomical or pathological evidence that is really valuable concerning the existence of a cortical centre for upward or downward associated movements, but in reasoning from analogy we must assume that such a centre or centres exist. The clinical case that Parinaud[29] reported in 1892 as indicating a lesion of the frontal centre, is in the light of our present knowledge unsatisfactory because of the association of homonymous lateral hemianopsia.

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[27] Neurologisches Centralblatt, Nov. 16, 1904, p. 1035.

[28] Revue Neurologique, Feb. 15, 1905, p. 161.

Parinaud reported the case before the Society of Ophthalmology, April, 5, 1892. The man had paralysis of the lower left part of the face, of the left half of the tongue, and temporarily of the left hand. He had almost complete paralysis of the elevators of the eyeballs, with a little reduction in the extent of the lateral movements. Convergence was well performed. (Here Parinaud presents a case of paralysis of upward associated movement with preservation of convergence.) Downward movement was normal. Parinaud believed there was a lesion in the right frontal lobe, at the level of the lower part of the Rolandic fissure, involving the centres for the face, tongue, hand, and associated movements of the eyeballs.

This patient had transitory partial left homonymous hemianopsia, and this was supposed to be the result of a lesion near the visual zone, which occupies, according to the teaching of Parinaud in 1892, all the occipital lobe, and extends to near the ascending parietal convolution. This opinion we now can hardly accept.

Hysteria may cause paralysis of the associated ocular movements, but there is no pathognomonic sign of this form of the palsy, and in some instances a correct diagnosis may be extremely difficult or even impossible. No case presents this difficulty more clearly than the one reported by Crouzon, Marie, and Babinski[30] before the Neurological Society of Paris on three different occasions in 1900 and 1901.

Crouzon presented the case as one of spasmodic tic, later Babinski and Parinaud expressed the opinion that it was organic and caused by a supranuclear lesion, and still later Marie presented the case with the diagnosis of a purely functional disorder, a neurosis, as a spasm of the elevators of the eyeballs. When the patient threw his head as far backward as possible and followed with his eyes a finger lowered slowly the eyeballs moved downward, but if his head were erect and he attempted on command to look at his feet, he bent his head forward and the eyeballs went forcibly upward. After thirty or forty seconds the spasm disappeared and the eyeballs returned to the normal position. Marie concluded from this that there must be a spasm of the elevators and not a paralysis of the depressors. The disorder had occurred after an apoplectic attack lasting seventeen hours, and the man had become blind. During the attack he had not had stertorous breathing nor passage of urine or fæces, and this Marie regarded as remarkable if there were an organic lesion. The coma disappeared suddenly, but the patient was delirious for several weeks and did not recognize anyone or his surroundings. This Marie regarded as like hysteria. The speech was slow and hesitating, and the visual fields were contracted.

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[29] Annales d'Oculistique, vol. cvii. p. 283, 1892.

[30] Revue Neurologique, 1901, p. 428.

Parinaud pointed out that the downward movement in following a finger is a reflex act and better performed than a voluntary movement, and this he says he has observed in hysteria, but when the condition is hysterical there is no inconvenience from the ocular disturbance, just as there is no inconvenience from the contraction of the visual fields in hysteria, and Parinaud has never seen a hysterical patient incline the head forward in order to make use of the superior part of the *champ du regard* (Dr. de Schweinitz suggests as a suitable translation for this term, field of fixation). The dissociation between the voluntary and reflex movements is not always hysterical. Parinaud, has pointed out that spasm may occur in sound ocular muscles as the result of paralysis of other ocular muscles when the patient is required to look in different directions. A paralysis confined to one eye may be the cause of spasm in the other, so that the sound muscles may appear to be affected. In these cases the paralysis is peripheral, but even in associated palsies it is not rare to see disturbance of the antagonistic muscles. In conjugate lateral paralysis from lesion of one sixth nucleus there is almost always nystagmus when the patient looks toward the sound side, and this nystagmus may be so pronounced that it may be difficult to say which side is affected. This I also have noticed in a case of complete paralysis of lateral associated movements on one side and of partial paralysis on the other. In the case which called forth so much discussion in the Neurological Society of Paris the visual fields were contracted only in the inferior portion and not concentrically, therefore the contraction was not like that of hysteria. The *champ visual* (visual field) Parinaud distinguishes from the *champ du regard* (field of fixation). He believes that if the cortical centre for associated movements of the eyes in the frontal lobe be destroyed, the voluntary associated movements will be lost, but the reflex associated movements will be preserved, and yet little, if any, clinical evidence in support of this opinion can be found.

Babinski pointed out that in the case under discussion the downward movement of the eyes was imperfect with the head thrown back. He quoted the case reported by Schröder[31] in which only paralysis of the associated downward movement persisted, and the eyes could not be lowered below the horizontal plane. Any attempt to look downward caused a spasm of the elevators and the eyes turned upward.

Oppenheim[32] has observed cases of pseudobulbar paralysis in which lateral movements of the eyeballs were impaired in voluntary innervation, but were preserved when the patient tried to follow an object or to turn toward the direction from which a sound came.

All the pathological evidence that I have been able to obtain in cases of persistent palsy of associated upward or downward movement is indicative of a lesion near the aqueduct of Sylvius. It is extremely doubtful whether a lesion confined to the corpora quadrigemina and causing no pressure on the surrounding parts ever causes paralysis of associated ocular movements, and those who favor such a view have not produced proof of a supranuclear centre in this part. Bernheimer[33] says Topolanski showed that electrical irritation of the corpora quadrigemina of rabbits does not produce ocular movements, and that these parts could be removed without producing symptoms. He believed that there is a centre for ocular movements near the oculomotor nucleus.

Bernheimer, from experiments on monkeys, has shown that the ocular movements do not depend on the integrity of the corpora quadrigemina, that this part of the brain does not contain a reflex centre for these movements, and that after destruction of this part the ocular movements are normal. He obtained synergic ocular movements from irritation of the angular gyrus both before and after he had removed the corpora quadrigemina. He thus refutes Prus's statements that a centre for ocular movements exists in the corpora quadrigemina.

Cases have been reported in which the corpora quadrigemina have been destroyed without disturbance in the movements of the eyeballs (Weinland, Seidel, Ruel, Nissen, cited by von Kornilow).

In a case that I have had the opportunity of studying there was a complete paralysis of right associated lateral movements, and great impairment of left and of upward associated movements. The paralysis of lateral associated movements is to be explained by the involvement of both posterior longitudinal bundles. If we assume that the nuclei of the superior rectus and inferior oblique muscles are in the posterior part of the oculomotor nucleus, we can understand why marked impairment of upward movement was present in this case, but this is contrary to the views held by some concerning the position of these groups of nerve cells.

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[31] Quoted by Teillais, Bul. de la Soc. Fran. d'Ophthal., 1899, p. 415.

[32] Neurologisches Centralblatt, 1895, p. 40.

[33] Wiener klin. Wochenschrift, No. 52, 1899, p. 1310.

Siemerling[34] has recently remarked that the numerous experimental studies of Bach, Bernheimer, van Gehuchten, and van Biervliet have not led to uniform conclusions. The oculomotor nucleus is merely the place for the transference of impulses and we are not in a position to state which parts are concerned with the innervation of the individual muscles.

Another of my cases is further evidence that the nuclei of the superior rectus and inferior oblique muscles may be in the posterior part of the oculomotor nucleus, because there was paresis of upward associated movement and the nerve-cells of the oculomotor nuclei were not diseased, but the posterior part of the nuclei was affected by pressure from the tumor in the pons, and the aqueduct of Sylvius was compressed upon the left side. The tumor did not invade the nuclei of the third and fourth nerves. The paralysis of left associated lateral movement is explained by the almost complete destruction of the left half of the pons by a tubercle, and thereby involvement of the posterior longitudinal bundle. The left sixth nerve was also much degenerated. In this and a similar case studied by me, downward movement was normal. This would seem to be evidence that the nucleus of the inferior rectus muscle must be in advance of those for the superior rectus and inferior oblique muscles, as in each case the tumor grew from the pons and did not invade the oculomotor nucleus.

If this is the correct position of these nuclei, a lesion developing ventrally in the pons as shown in two of my cases may by pressure upon the posterior part of the oculomotor nuclei cause paralysis of associated upward movement, as a result of injury of the nuclei for the superior rectus and inferior oblique muscles, and it would not be at all necessary to assume the existence of a co-ordinating centre for upward associated movement. Paralysis of downward associated movement depends on impairment of the inferior rectus and superior oblique muscles, these having cells of origin in two distinct nuclei. There must be a close association between the nucleus of the superior oblique and that of the inferior rectus muscle of the same side, and possibly of the opposite side. It is probable therefore that a lesion of the nuclei of the inferior rectus muscles and of the fibres connecting them with the nuclei of the trochlear nerves would cause paralysis of associated downward movement, and it seems unnecessary to assume that a co-ordinating supranuclear centre for upward or downward movements exists in the corpora quadrigemina or their vicinity. Indeed, two of my cases show that such a centre is improbable, because in each case the lesion causing paresis of upward associated movement was posterior to the oculomotor nucleus, and did not implicate the corpora quadrigemina. Three of the cases of the series studied by me afford further evidence against a centre for co-ordinate ocular movements in the corpora quadrigemina. In one the paralysis at first was only in upward movements, later also in lateral associated and in downward associated ocular movements. In this case, therefore, a paralysis at first confined to one form of associated movement later extended to other forms. In another case the paralysis of upward associated ocular movements disappeared, leaving only paresis of the left superior rectus, and finally this also disappeared. In another case the associated palsy disappeared, leaving paralysis of muscles only on one side. It is very common to have a palsy of one or more branches of the oculomotor nerve in association with paralysis of associated upward or downward movement, and this is more easily explained by a lesion of the oculomotor nucleus than by a cortical lesion. Cortical centres control associated movements, not isolated muscles.

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[34] Archiv. für Psychiatrie, vol. xl. No. 1, p. 61.

The paralysis of the sixth nerve occurring with paralysis of associated upward or downward movements has been attributed by Lichtheim to pressure upon the nerve at the base of the brain. The opinion held by Parinaud, although supported by Duval, Laborde, and Graux, can hardly be accepted,—*viz.*, that the sixth nucleus gives nerve fibres to the internal rectus muscle, and that this muscle has a double innervation, the sixth nerve for lateral movement, the third for convergence.

The assumption of a basal co-ordinating centre above the oculomotor nucleus for ocular movements seems forced. The lateral associated ocular movements can be well explained by the connection formed by the posterior longitudinal bundle between the nucleus of the branch to the internal rectus muscle and the nucleus of the sixth nerve. It is reasonable to suppose that certain fibres connecting the different parts of the oculomotor nucleus with one another, or connecting the group of nerve cells innervating the inferior rectus with the nucleus of the trochlear nerve has a co-ordinating function similar to that of the posterior longitudinal bundle, and that this function may be disturbed by a small lesion. It is striking that the paralysis of downward associated movement (necessitating the implication of two separate nuclei of ocular muscles) without paralysis of upward associated movement is exceedingly rare, far more so than is the isolated paralysis of upward associated movement.

Conjugate deviation of the eyeballs from lesions of the pons is not common when the lesion is confined to one side of the pons. In a case of hemorrhage into the tegmentum of the pons that I have had the opportunity to study and the brain from which I have examined, inward deviation of only the right eye occurred from paralysis of the sixth nerve. One gets the impression that conjugate deviation of the eyeballs away from the side of the lesion is common in lesions of one side of the pons, but in four cases of lesions of this part observed by me (three of tumor and one of hemorrhage) causing paralysis of the external rectus muscle of the same side, I have looked for the conjugate deviation, but failed to find it. It is probable that in most cases the side of the pons opposite to the lesion is directly or indirectly involved.

Von Monakow[35] believes that when the lesion implicates the sixth nucleus of one side, apart from the paralysis no disturbance,—*i.e.*, no deviation occurs, unless it is the result of secondary contracture. I believe that conjugate deviation is rare even as a late sign of paralysis of one sixth nucleus. Of course any involvement of both sixth nuclei would make conjugate deviation impossible, and in one of my cases both these nuclei were affected.

In a case of softening of the dorsal part of the left side of the pons reported by Albert Ransohoff[36] there was complete paralysis of the associated ocular movement to the left and of convergence movement of the right eye. The right external rectus muscle was not overactive, but the left internal muscle was in marked spasm. The left abducens nucleus was involved in the lesion, the right escaped, but the right posterior longitudinal bundle was involved. Ransohoff mentions that Wernicke and Jolly have shown that paralysis of the external rectus muscle does not in itself cause contraction of its antagonist, the internal rectus, and that in the cases of these authors, as well as in his own, there was a lesion of the opposite posterior longitudinal bundle. In Wernicke's case a tumor had destroyed the left adducens nucleus and invaded the right side of the pons. Both eyeballs were persistently turned to the right, especially the left eyeball.

According to the study of the literature by Ransohoff, contracture of the unparalyzed antagonistic muscles, but not always in both eyes, and more in the internal rectus muscle on the side of the lesion than in the external rectus muscle on the side opposite to the lesion, was observed in the cases of Wernicke, Jolly, Graux, Meyer (the lesion did not extend to the mid-line), Spitzer (slight, and after one-half year duration of the lesion), Mierzejewsky and Rosenbach, Gebhard, and Ransohoff.

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[35] Gehirnpathologique, p. 335.

[36] Archiv. für Psychiatrie, vol. xxxv. 1901, p. 416.

Ransohoff says that if the posterior longitudinal bundle, as is assumed by most authors, contains a part of the voluntary fibres for the internal rectus muscle of the opposite side, a lesion of these fibres, as of the pyramidal fibres, must cause spastic contracture of the muscle they innervate. Therefore, according to Ransohoff, contracture of the internal rectus muscle on the side of the lesion indicates that the posterior longitudinal bundle of the opposite side is also involved, but this contracture probably could not exist if the nucleus of each abducens were destroyed.

In four of my cases both posterior longitudinal bundles must have been much affected and conjugate deviation did not occur in any.

In one case in which paralysis of lateral movements was present, the movement of either eyeball to the right or left was distinctly greater when one eye was covered than in associated lateral movements. The individual movements of the eyeballs were therefore greater than the associated movements.

One of my cases was especially interesting in that syphilitic infection was positive. The paralysis of associated movement was peculiar; the downward movement was completely lost in the right eye and much impaired in the left; the upward movement was slight in the right eye and completely lost in the left; the lateral associated movements were nearly normal. Almost all the ocular movements were regained except the downward movement of the right eye. It is remarkable that the complete paralysis in the associated movements was in the downward direction in one eye and in the upward direction in the other, although the associated movement was very feeble. It is remarkable that the associated palsy disappeared leaving paralysis of downward movement of the right eye.

In four cases of my series with necropsy the pupil was smaller on the same side as the lesion of the pons. This alteration of the size of the pupil from a lesion of the pons is difficult to explain, but the presence of the sign in four cases shows that it is of diagnostic significance, and also that the fibres controlling the iris pass through the pons.

In at least two cases nystagmus was present in looking away from the side of the chief lesion. This was caused by weakness of the opposite external rectus muscle from pressure upon or direct involvement of the nucleus of the abducent nerve. This sign may be expected when a lesion affects the dorsal part of the pons and destroys the function of one abducent nucleus and weakens that of the other. Nystagmus may surely be the result of paresis of ocular muscles.

I have studied thirty-eight cases of paralysis of upward or downward associated movement reported in the literature, not including nine of my own. In many cases certain symptoms are not referred to, so that statements regarding the frequency of symptoms are incomplete. Von Kornilow used only twenty cases of the twenty-seven to which he found references in drawing his conclusions. As a result of my study of these forty-seven cases,—

Paralysis of upward associated movement without paralysis or paresis of downward associated movement was found in twenty-six cases (Henoch, Priestley Smith (2), Nieden, Gowers, Parinaud (2), Ormerod, Reinhold, Thomsen, Lichtheim (3), Hoesslin, Bruner, Mills, Noguès and Sirol, Kornilow, Posey, Parkinson, Cases 1, 2, 4, 5, 6, 8 of my series).

Paralysis of upward associated movement without paralysis or paresis of downward associated movement was found in sixteen cases (Wernicke, Parinaud, Hope, Nothnagel, Eisenlohr, Verrey, Sharkey, Sauvineau, Teillais, Gordinier, Raymond and Cestan (slight), Kornilow, Cases 3, 4, 7, 9 of my series).

Paralysis of upward associated movement with impairment of lateral movement, often developing later, was found in fifteen cases (Henoch, Priestley Smith (2), Lichtheim (3), Parinaud, Poulard, Raymond and Cestan, Kornilow, Parkinson, Cases 1, 2, 3, and 4 of my series).

Paralysis of upward associated movement without impairment of lateral movement was found in twenty-two cases (Wernicke, Nieden, Gowers, Parinaud (2), Ormerod, Hope, Thomsen, Sharkey, Sauvineau, Hoesslin, Bruner, Teillais, Gordinier, Noguès and Sirol, Kornilow, Posey, Cases 5, 6, 7, 8, and 9 of my series).

Paralysis of downward associated movement without paralysis of upward associated movement was found in five cases (Gee, Poulard, Curzon-Marie and Babinski, Basevi, Schröder), but in none of my nine cases.

The reaction of the iris was found to be impaired in fourteen cases (Henoch, Gowers, Parinaud, Thomsen, Eisenlohr, Lichtheim, Sharkey, Sauvineau, Hoesslin, Gordinier, Mills, Kornilow, Posey, Case 9 of my series), and is said to have been normal in four cases (Parinaud, Teillais, Poulard, Raymond and Cestan), but in many cases no statements regarding the iritic reflex are made, and in all my nine cases the iritic reflex was preserved.

The optic nerve was found to be affected in fifteen cases (Gowers, Thomsen, Nothnagel, Gee, Eisenlohr, Lichtheim (3), Sharkey, Hoesslin, Gordinier, Cases 1, 5, 7, and 9 of my series), and normal in twelve cases (Wernicke, Nieden, Parinaud (2), Ormerod, Reinhold, Sauvineau, Teillais, Raymond and Cestan, Mills, Nogués and Sirol, Posey).

Convergence was impaired in fifteen cases (Parinaud (2), Sharkey, Sauvineau, Teillais, Poulard, Gordinier, Raymond and Cestan, Kornilow, Basevi, Cases 2, 4, 5, 8, and 9 of my series), and normal, or nearly normal, in nine cases (Hope, Parinaud, Nogués and Sirol, Kornilow, Posey, Cases 1, 3, 6, and 7 of my series).

Ptosis was found in seven cases (Reinhold, Eisenlohr, Gordinier, Kornilow, Nothnagel, Cases 2 and 8 of my series), and was said to be absent in thirteen cases (Wernicke, Parinaud, Gee, Sauvineau, Teillais, Raymond and Cestan, Cases 1, 3, 4, 5, 6, 7, and 9 of my series).

Necropsy was obtained in nineteen cases (Henoch, Wernicke, Gowers, Hope, Reinhold, Thomsen, Gee, Eisenlohr, Lichtheim (3), Sharkey, Hoesslin, Gordinier, Nothnagel, Basevi, Cases 1, 2, and 3 of my series).

In all of these cases except one (Thomsen[37]) parts about the aqueduct of Sylvius were implicated, and in Thomsen's case a gumma was found in the cerebral peduncles. Thomsen cannot explain why the intense degeneration of the right oculomotor nerve and the slight degeneration of the left oculomotor nerve caused paralysis of only upward movement. It is possible that the Nissl method, if it had been employed, would have shown marked alteration of the oculomotor nuclei, possibly as a toxic effect from the adjacent gumma, and in this way the paralysis of upward associated movement may have been caused. Such was the conditon in one of my cases, unless perhaps the alteration of the nerve cells was caused by pressure. Until at least another case similar to Thomsen's is reported we may well hesitate to believe that a peripheral lesion of one oculomotor nerve may cause paralysis of only upward associated movements.

Tumor was found in fourteen cases (Henoch, Gowers, Hope, Reinhold, Gee, Lichtheim (3), Sharkey, Hoesslin, Gordinier, Nothnagel, Cases 1 and 2 of my series), a bullet wound in one case (Eisenlohr), apoplectic cyst in one case (Wernicke), hemorrhage in one case (my Case 3), and lesion uncertain in one case (Basevi).

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[37] Archiv. für Psychiatrie, vol. xviii. 1887, p. 616.

Recovery or partial recovery occurred in seven cases (Priestley Smith, Nieden, Verrey, Kornilow, Cases 5, 8, and 9 of my series). Important symptoms other than ocular palsies were found in forty-one cases (Henoch, Wernicke, Priestley Smith (2), Gowers, Parinaud (3), Hope, Reinhold, Thomsen, Nothnagel, Gee, Eisenlohr, Lichtheim (3), Verrey, Sharkey, Sauvineau, Hoesslin, Bruner, Teillais, Poulard, Gordinier, Cruzon-Marie and Babinski, Raymond and Cestan, Mills, Nogues and Sirol, Kornilow (2), Schröder, Posey, Parkinson, Cases 1, 2, 3, 4, 6, 8, and 9 of my series).

As a result of my studies I believe that persisting paralysis of associated lateral movement indicates a lesion of the posterior longitudinal bundle; that persistent paralysis of associated upward or downward movement indicates a lesion in the vicinity of the oculomotor nucleus, and that paralysis of associated ocular movements is not the result of a lesion of extracerebral nerve fibres. Lesions of the cerebral cortex may certainly cause paralysis of lateral associated ocular movements, and possibly of upward or downward associated ocular movements, but cortical paralysis of associated ocular movements is transitory, unless possibly where the centre on each side of the brain is destroyed. Paralysis of associated ocular movements may be caused by hysteria. Any case in which associated ocular palsy is persistent and is of organic nature, is unsuitable for operation unless the operation is merely palliative, as the lesion is probably within the posterior part of the pons or cerebral peduncle, according to the form of the associated palsy, or else causes much pressure upon the dorsal portions of these structures. The paralysis of associated ocular muscles may be produced by inflammatory lesions or lesions of a similar character (alcohol, syphilis), as well as by tumor, and may disappear later in the course of the disease. Syphilitic ependymitis and cellular infiltration must be considered in diagnosing the lesion causing paralysis of associated ocular movements. Most congenital associated palsies are probably nuclear in origin.

Drowsiness, Nystagmus, and Impairment of Vision as Signs of Tumor of the Corpora Quadrigemina.—Drowsiness and repeated yawning are not uncommon signs of a tumor of the corpora quadrigemina, but are not pathognomonic.

Nystagmus also occurs with tumor of the corpora quadrigemina and sometimes is obtained only by looking upward.

A peculiar form of nystagmus is described by Bielschowsky as occurring in cases of tumor of the corpora quadrigemina. It consists of clonic movements of both internal rectus muscles, and seems to have been much the same form of movement as that observed by Ransom.

Bielschowsky refers to Blanquinque as having described in 1871 convulsive movements of the eyeballs as a sign of tumor of the corpora quadrigemina.

Vision may not be disturbed at all by a tumor of the corpora quadrigemina, and when disturbed vision results from optic neuritis the latter probably is more frequently caused by internal hydrocephalus. Implication of the anterior colliculi of the corpora quadrigemina probably does not cause marked disturbance of vision, but the external geniculate body of one side may be involved and give rise to hemianopsia if the tumor grows forward.

In a case of tubercle of the corpora quadrigemina with tuberculous meningitis, reported by Nissen, the failure of vision seemed to be caused by the optic neuritis from meningitis, and not by the tubercle of the corpora quadrigemina.

In W. Goldzieher's case of tubercle of the corpora quadrigemina the symptoms were: ataxic gait, drowsiness, complete paralysis of all external muscles supplied by each oculomotor nerve (the movements of the iris were normal and accommodation also seemed to be normal, as the child recognized small objects held near its eyes; the eye-grounds were normal), weakness of the lower limbs, frequent yawning, sudden and severe involuntary jerkings of the whole body, etc. The child was five years old.

The necropsy showed a tubercle of the corpora quadrigemina about the size of a hazel nut. The aqueduct of Sylvius was somewhat narrowed by pressure, but the gray matter about the ventricles did not appear to be implicated.

The case shows, according to Goldzieher, that a tumor involving the corpora quadrigemina entirely may cause no blindness. He refers to Nothnagel as having spoken of this fact in his Topischen Diagnostik der Gehirnkrankheiten, pp. 211–214. Goldzieher's patient saw well, and accommodated.

In Bielschowsky's case the anterior portion of the corpora quadrigemina was completely degenerated, and each pulvinar was much compressed, but vision was only a little impaired, and the visual fields were not contracted. The external geniculate bodies were preserved, and to the integrity of these structures was due the preservation of vision.

In some cases of tumor of the corpora quadrigemina the abducens nerve has been paralyzed, as in a case observed by W. B. Ransom,[38] in which there was a large oval-celled sarcoma of the corpora quadrigemina.

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[38] Lancet, May 4, 1895, p. 1115.

Ophthalmoplegia was well developed long before marked ataxia set in. In the earlier stage the diagnosis between growth from the base of the skull, of the corpora quadrigemina or pineal gland, and of the cerebellum, was not certain. Paralysis of the sixth nerves, loss of knee-jerks, clonic spasm of the upper lids and internal rectus muscles, especially of the left, were observed. The disturbance of vision in this case was regarded by Ransom as unusual, as in most cases such loss has been slight or very late in appearing. It could not in his case have been due, he says, to implication of the optic thalamus or optic radiations. It is possible that the optic neuritis caused it, but a similar amount of effusion is frequently seen with little or no loss of vision. The paralysis of each external rectus is interesting as the pons was normal. An explanation for this paralysis is not given, but the case is another instance of the paralysis of the abducent nerve when the lesion is at a distance from its nucleus.

Optic neuritis was absent in a case of tumor in the corpora quadrigemina observed by Frederick Taylor,[39] in which the cerebellum was perfectly healthy, but the corpora quadrigemina were flattened, broader, and gray and gelatinous in appearance, and were the seat of a new growth, regarded as a glio-sarcoma. The symptoms were: ptosis, staggering gait, drowsiness, ataxia of the upper limbs, nearly complete double ophthalmoplegia, lateral nystagmus and later unconsciousness. The pupils were slightly and unequally dilated and reacted to light, but the child could not see.

Nissen [40] has made the peculiar observation that tumor of the brain in children is less likely to cause choked disks than is tumor in adults. In his five cases of tumor of the corpora quadrigemina or cerebral peduncle, optic neuritis was present only in one, and in this case there was meningitis. Nissen mentions as cases of tumor of the corpora quadrigemina with normal eyegrounds those of Taylor, Goldzieher, Bruns, and Ilberg.

Resemblance between the Symptoms of Tumor of the Corpora Quadrigemina and those of Multiple Sclerosis.—The symptoms of tumor of the corpora quadrigemina may resemble closely those of multiple sclerosis, and the differential diagnosis may be exceedingly difficult. For example, in Bielschowsky's case [41] the temporal side of each disk was pale as in multiple sclerosis. This pallor was explained as secondary atrophy from pressure on the chiasm by the distended third ventricle. Pressure on the chiasm may cause atrophy without first producing choked disks. This temporal pallor of the disks is confusing, especially in cases of tumor of the corpora quadrigemina, as Bielschowsky points out, because incoördination is also present, and the diagnosis of multiple sclerosis is likely to be made.

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[39] Taylor, Lancet, November 18, 1893, vol. ii. p. 1252.

[40] W. Nissen Jahrbuch für Kinderheilkunde, 1901, vol. liv.

[41] Bielschowsky, Deutsche Zeitschrift für Nervenheilkunde, xxii. 1 and 2, p. 54.

Nystagmus and coarse intention tremor in Gordinier's case were suggestive of multiple sclerosis, but Gordinier believed that severe headache, optic neuritis, ophthalmoplegia, and the cerebellar gait, together with the absence of scanning speech, of epileptiform attacks, and of evidence of motor-tract involvement, negatived such a diagnosis. This opinion can not be accepted unconditionally. To my mind the absence of involvement of the motor tract was the best clinical evidence that the disease was not multiple sclerosis. Headache, optic neuritis, cere-

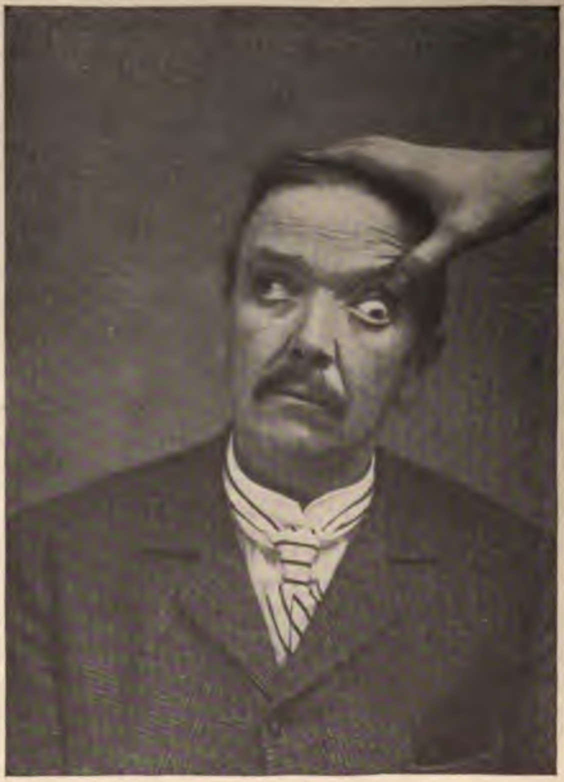

FIG. 11.—Tumor of orbit causing ptosis of left upper eyelid, almost immobile pupil, complete paralysis of superior rectus and inferior rectus, partial paralysis of internal rectus, some protrusion of eyeball, and blurring of optic disk on the left side. The photograph shows an attempt to look to the right. The only sign of intracranial disturbance was exaggeration of the patellar reflexes. Had the tumor grown more within the cranium, the limbs of the right side would have been paralyzed in association with the left oculomotor paralysis.

bellar gait may all occur in multiple sclerosis, and I have shown by a case with necropsy that ophthalmoplegia may also be a sign of the disease. Scanning speech and epileptiform attacks are not present in every case of multiple sclerosis.

Symptoms from Implication of the Cerebral Peduncle.—When the tumor extends far enough ventrally to involve the foot of the cerebral peduncle it may cause paresis or paralysis of the limbs on the side opposite to the lesion, with involvement of the oculomotor nerve on the same side as the lesion. Various forms of tremor may be present in the paretic limbs, the involuntary movements may have the characteristics of intention tremor, or may be choreiform or athetotic.

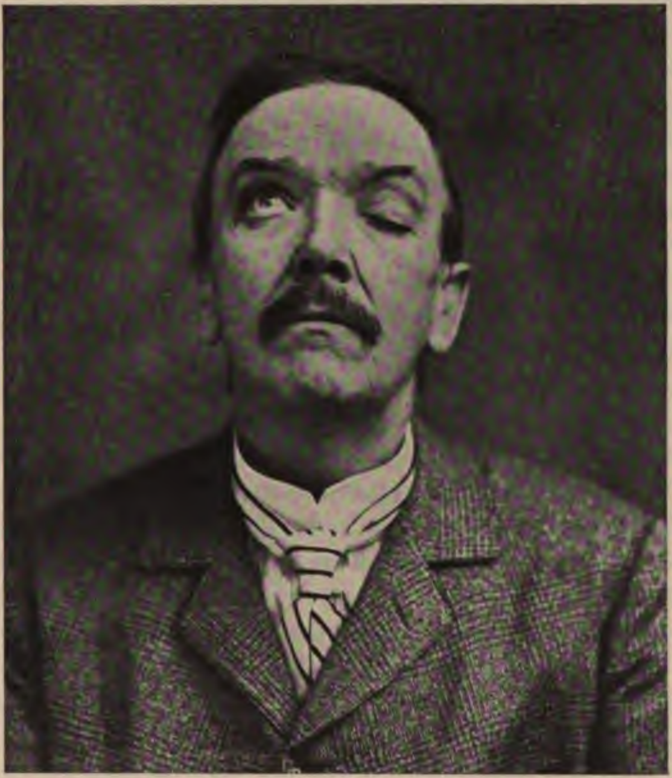

FIG. 12.—Same patient as shown in Fig. 11. Complete left-sided ptosis. (Case seen by author with Dr. S. D. Risley.)

In a case that B. Sachs [42] reports, one of thrombosis of the posterior cerebral artery, the right crus was small and very soft, and the right oculomotor nerve was as thin as a thread. Left hemiplegia had been present. The left upper limb had shown the wildest ataxic movements, reminding one of the extreme incoördination of the lower limbs in the last stages of tabes dorsalis. If the patient were asked to use the paralyzed hand the paretic limb was whirled about so violently as to be a source of some danger to those near. After a little, the arm would fall down from exhaustion, and remain quiet until aroused again.

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[42] Sachs, Amer. Jour. of the Med. Sci., 1891, vol. ci. p. 233.

In a case observed by Bristowe,[43] all the limbs were weak but not paralyzed, and were tremulous on movement; this, according to Bristowe, seemed to indicate compression and irritation of the motor tracts.

In another case a tuberculous growth occupied the third ventricle and implicated both optic thalami. Tremors like those of disseminated sclerosis and drowsiness were present in this case.

Kolisch [44] says (1893) that ataxic gait has been observed in two-thirds of the cases of tumor of the corpora quadrigemina. Peculiar persisting choreiform movements of the left upper and lower extremities, but ceasing during sleep, were observed even when his patient, a child, was lying on her back, and were more intense when she was excited, sat up, or attempted to walk. These movements were attributed by Kolisch in his case to lesion of the tegmentum of the cerebral peduncle and irritation of the pyramidal tract.

Gordinier says that in five of the eleven cases in literature (Bruns, Weinland, Eisenlohr, Ilberg, Bonhoffer) in which lesions of the corpora quadrigeminal region were accompanied by tremor or choreiform movements, the superior cerebellar peduncles were involved. Both Bruns and Weinland attribute the tremor to irritation of the motor fibres of the pyramidal tracts, although, as Gordinier mentions, in the cases they report the necropsies showed the motor tracts to be normal, while the superior cerebellar peduncles were diseased. Gordinier refers to the experiments of Ferrier and Turner, who described similar tremors resulting from division of the superior cerebellar peduncles in monkeys. They found that if a peduncle were divided between the cerebellum and its decussation in the tegmentum, the tremor was confined to the side of the lesion. I have referred in my section on tumors of the optic thalamus to involuntary movements caused by implication of the superior cerebellar peduncles.

Symptom-Complex of Benedickt.—Paralysis of the oculomotor nerve on one side and of the limbs on the opposite side, with tremor of any variety, has been called the symptom-complex of Benedickt, but is hardly to be expected in a typical form in tumor of the corpora quadrigemina, as the ocular nuclei are situated so close together that the paralysis of the ocular muscles is usually bilateral, although not commonly symmetrical. In order to have paralysis of the limbs the tumor must in some way affect the motor fibres in the foot of the cerebral peduncle.

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[43] Bristowe, Brain, vol. vi. p. 167.

[44] R. Kolich, Deutsche Zeitschrift für Nervenheilkunde, vol. iv. p. 14.

In a very interesting case of tubercle of the right cerebral peduncle, reported by Blocq and Marinesco,[45] there were symptoms like those of paralysis agitans. The diagnosis of paralysis agitans was made during the life of the patient. The immobile face was like that occurring in this disease. A rhythmical tremor of small oscillations was seen in the left hand, the left fingers were half flexed, the left lower limb trembled also but less severely. Voluntary effort arrested the tremor temporarily. The authors believe that this was not paralysis agitans occurring in a case of tumor of the cerebral peduncle, because of the exaggeration of the reflexes on the left side and the exact correlation of the symptoms with the localization of the tumor. Exaggeration of tendon reflexes, however, does occur in paralysis agitans.

They refer to a case of Mendel in which intention tremor of the right arm was found associated with a tubercle of the left cerebral peduncle. Charcot observed a case in which tremor like that of Parkinson's disease, with typical attitude of one of the upper limbs, was associated with a tumor compressing one of the cerebral peduncles.

Deafness as a Sign of Lesion of the Corpora Quadrigemina.—Weinland[46] has made an important study concerning the effect of lesion of the posterior colliculi of the corpora quadrigemina upon hearing. In nineteen cases of tumor of the corpora quadrigemina disturbance of hearing was observed in nine cases,—*i.e.*, in about half of the cases (Duffin, Klebs-Fischel, Gowers, Ferrier, Fischer, Hope, Nothnagel, Ruel, Weinland). Disturbance of hearing was observed in four cases out of nine of some other form of lesion of the corpora quadrigemina (Hintz, Reinhold, Henschen, Zenner). In five of the nine cases of tumor of this region, with disturbance of hearing, the deafness was bilateral, although not always equal on the two sides. In three cases (Ferrier, Ruel, Weinland) it was shown that diseases of one posterior colliculus causes disturbance of hearing on the opposite side. This is explained by the course of the fibres concerned in hearing. Fibres connecting with the cochlear nerve form the lateral fillet, which passes to the posterior colliculi of the corpora quadrigemina; from here other fibres pass to the internal geniculate body and the first and possibly the second temporal convolutions.

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[45] Blocq and Marinesco, Mémories de la Soc. de Biologie, 1893, p. 105.

[46] Weinland, Archiv. für Psych., 1894, vol. xxvi. p. 353.

Weinland shows that the cases of tumor of the corpora quadrigemina, in which deafness was not observed, do not lessen the importance of the posterior colliculi in the function of hearing.

In Gordinier's case[47] hearing was normal although the posterior colliculi of the corpora quadrigemina were almost destroyed. The patient heard ordinary conversation distinctly and the watch three feet away from each ear. This was explained by the absence of implication of the lateral fillet of either side or of the nucleus about which its fibres terminate. In Bielschowsky's case also the posterior colliculi of the corpora quadrigemina were much compressed, and yet hearing was not positively affected thereby, so far as could be determined, although the patient felt as though the hearing had become less acute.

Diagnosis between Lesions of the Corpora Quadrigemina and those of the Cerebellum.—Extension of the tumor forward so as to involve the visual system, either the optic tract, external geniculate body, or the optic thalamus, causes homonymous lateral hemianopsia. Extension backward into the pons may result in paralysis of the motor and sensory portions of the trigeminal nerve, and of the facial and abducens.

L. Bruns[48] emphasizes the difficulty in diagnosing between tumors of the corpora quadrigemina and those of the cerebellum, as ataxia and ophthalmoplegia may be caused by a tumor in either location. A tumor of the vermis is very likely to cause cerebellar ataxia, and by pressure upon the corpora quadrigemina may cause ocular palsies.

He believes on theoretical grounds it is possible that a tumor elsewhere within the brain, for example in the frontal lobe, may cause ataxia with paralysis of ocular muscles. Where the symptoms begin with ataxia Bruns thinks the tumor is probably in the cerebellum; where they begin with ophthalmoplegia the tumor is probably in the corpora quadrigemina. This is entirely contrary to Nothnagel's teaching, as the latter regards ataxia occurring as the first symptom before the ophthalmoplegia as especially diagnostic of tumor of the corpora quadrigemina. Bruns mentions as a diagnostic point that the escape of the abducent nerves would be more in favor of tumor of the corpora quadrigemina than of tumor of the cerebellum, but paralysis of these nerves may occur with tumor in either place. Implication of other cranial nerves, as of the facial, would indicate rather tumor of the vermis. Ataxia or choreiform movements would indicate rather tumor of the corpora quadrigemina.

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[47] Gordinier, Journal of Nervous and Mental Disease, October, 1901, p. 543.

[48] Bruns, Die Geschwülste des Nervensystems, Archiv. für Psych., 1894, vol. xxvi. p. 299.

Nissen says that Nothnagel believed the posterior colliculi of the corpora quadrigemina are concerned in co-ordination. Others believe the ataxia is caused by involvement of the red nucleus or superior cerebellar peduncle; still others attribute the ataxia to pressure on the cerebellum. In Nissen's case there was no ataxia and a tubercle was confined to the corpora quadrigemina and implicated only the upper part of the tegmentum. There was also no disturbance of hearing, although the posterior colliculi of the corpora quadrigemina were implicated. Nissen thinks that the corpora quadrigemina may be a reflex area, in which the acoustic system is connected with the nuclei for ocular movements. He thinks also that at least in some of the cases of tumor of the posterior colliculi with deafness the tumor may have pressed upon the internal geniculate body, or may have caused the deafness by distant pressure upon the medulla oblongata and acoustic nerve.

In this remarkable case of Nissen there was neither ataxia of limbs nor paralysis of the ocular nerves, and Nissen shows that the symptoms recognized by Nothnagel (ataxic gait and ophthalmoplegia) depend upon implication of the tegmentum.

In two cases of tumor of the corpora quadrigemina observed by W. Aldren Turner,[49] staggering gait combined with diplopia and headache varying in position were the chief symptoms. In one case the inclination was invariably backward, so that the patient was unable to stand or even to sit up in bed without support from behind. The ocular symptoms consisted in paresis of the third and sixth nerves in one case, and defect in the upward and downward movements of the globes in the other. Optic neuritis was present in both cases. In one case the knee-jerks were lost and in the other they were diminished. Both of these cases showed ataxia of cerebellar character with impairment in the movements of the eyeballs.

Persisting subnormal temperature was observed in a case of glioma of the mid-brain and left optic thalmus reported by Lloyd.[50] The paralysis was crossed, the left third nerve being involved, with right brachial monoplegia and right crural monoparesis. The patient had a remarkable course of subnormal temperature, the lowest point reached being 95.4°. The temperature was subnormal for twenty days, with two exceptions.

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[49] Turner, Brain, 1898, vol. xxi.

[50] Lloyd, Med. News, January 30, 1892, p. 129.

crossed fibres. A lesion on the outer side of each optic nerve near the chiasm, or on the outer side of each optic tract, might cause binasal hemianopsia. This subject has recently been carefully studied by W. T. Shoemaker.[53]

The definition of hemianopsia he gives is "Half defect in the field of vision of each eye, symmetrically placed, and caused by a common intracranial lesion." He believes that binasal hemianopsia can never result from a lesion of the chiasm on account of the anatomical arrangement of the fibres, and that in most instances it is a symptom of optic nerve disease. In eighteen cases reported in the literature and in one of his own of binasal hemianopsia demonstrable inflammatory disease of the optic nerves was found in twelve cases. Shoemaker refers to five cases which seem to have exhibited true binasal hemianopsia (Lang and Beevor, Graefe, Daae, Herschel, Mooren's second case).

Tumor growing from the pituitary body sometimes causes intense drowsiness. It was present in the case of acromegaly reported by A. F. Witmer from my clinic and also in the case of the same disease reported by F. A. Packard. F. W. Mott[54] says that in three cases of tumor of the third ventricle, which started probably from the pituitary body, and in which during life symptoms of such a growth were present, a condition of drowsiness was observed and was so intense that these patients would fall asleep while eating their meals, and in two of the cases this phenomenon was so marked a feature as to be the most prominent and early objective symptom.

Tumor in this situation I believe also may cause frequent yawning, but I have seen this sign when the tumor was in the cerebellum.

I have seen the entire third ventricle filled by a tumor growing from the pituitary body in a case in which the symptoms of acromegaly were pronounced (case of F. A. Packard, Fig. 13). Other illustrations of Dr. Packard's case are given in Dr. Burr's article.

TUMORS OF THE OPTIC NERVE.

Sometimes a tumor occurs in the intracranial portion of the optic nerves, as in a remarkable case reported by A. Pick.[55] Braunschweig collected cases of tumor of the optic nerves and concluded that exophthalmos was never absent in such cases. Pick shows by the report of his own case that the word "never" should have been omitted. At the

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[53] Shoemaker, New York Med. Journal and Philadelphia Med. Journal February 4, 1905.

[54] Mott, Brain, 1898, vol. xxi.

[55] A Pick, Brain, 1901, vol. xxiv. p. 502.

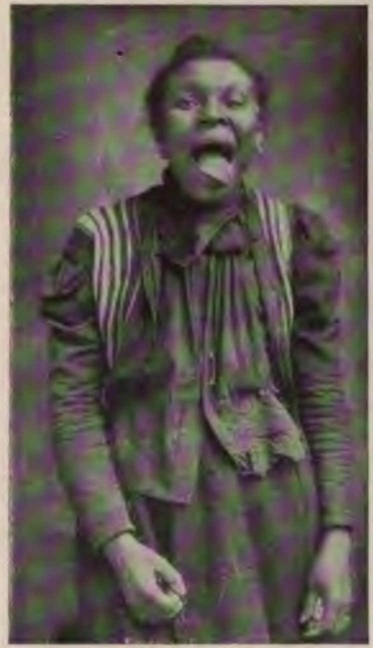

FIG. 14.—Paralysis of right upper and lower limbs and of right side of face; paralysis of left muscles of mastication and of left side of tongue. Tongue deviated to the left when protruded. Diplopia in looking to the left. A lesion of the pons was probably present in this case.

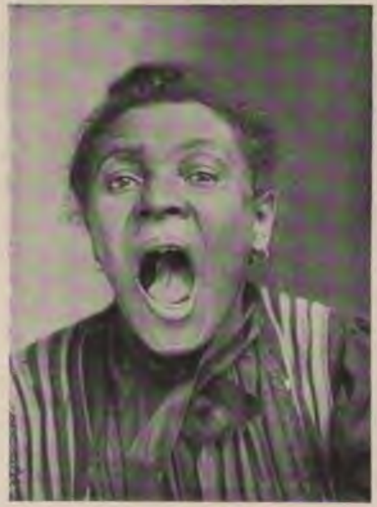

FIG. 15.—Same case as represented in Fig. 14. The lower jaw deviated toward the left when the mouth was opened because of the paralysis of the left muscles of mastication. The tongue deviated toward the right when within the mouth. Paralysis of left side of tongue.

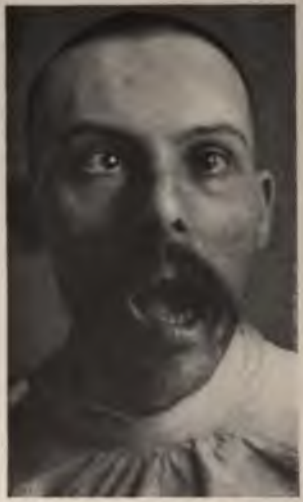

FIG. 16.—Paralysis of each external rectus and of the left muscles of mastication from a tubercle in the left half of the pons.

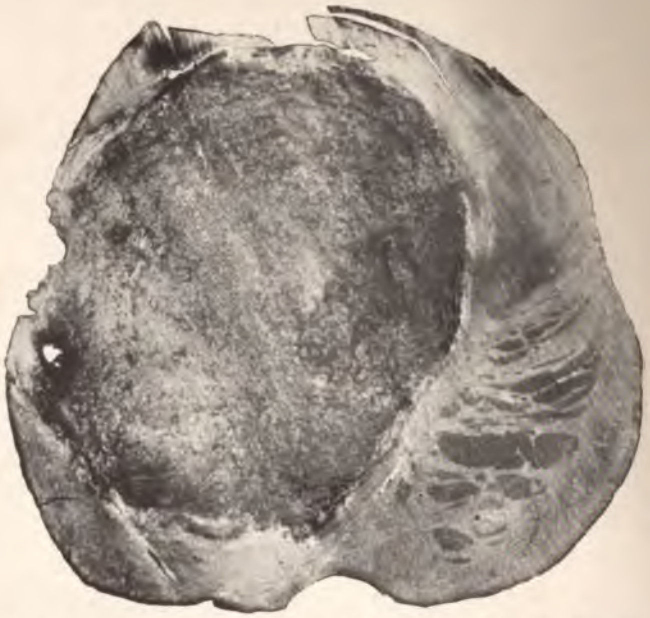

FIG. 17.—Same case as shown in Fig. 16. Tubercle of the left half of the pons.

necropsy in Pick's case the optic nerves appeared very gray, but were not diminished in size. The optic nerves, chiasm, and stump of the optic tract did not stain well by the Weigert-Pal method, and on superficial examination seemed to be merely atrophied; but more careful examination showed numerous cells among the atrophied nerve-fibres. The tumor was a myxosarcoma. Early failure of vision is to be expected in tumors of the optic nerve, and the reflex pupillary response may be present at one time and absent at another. The latter sign may not always be present, and is caused probably, as Pick says, by the soft tumor tissue within the nerve and by varying degrees of vascular congestion. Pick's patient had hallucinations of sight, similar to those sometimes occurring in disease of the retina or optic nerves.

TUMORS OF THE PONS AND MEDULLA OBLONGATA.

Ataxia is generally very pronounced in tumors of the pons, because of implication of the cerebellar peduncles, but I have had a case in which a large tumor on the pons caused no ataxia. Many important structures are crowded together in the pons, and even a small growth may cause very striking symptoms. When the tumor is confined to one side of the pons, it may cause paralysis of motion in the muscles of mastication and loss of sensation in the face on the same side as the tumor, because of involvement of the trigeminal nerve; paralysis of the external rectus and of the muscles of the face on the same side as the tumor, because of involvement of the abducent and facial nerves; in some cases also when the tumor extends low into the pons, deafness on the same side from involvement of the acoustic nerve, or of the intrapontile fibres belonging to the acoustic system. The tongue and limbs may be paralyzed on the opposite side because of implication of the central motor-fibres for the hypoglossus nucleus, and of those for the anterior horns of the cervical and lumbar region above their decussation. Sensation may be impaired in the limbs of the opposite side of the body and even in the opposite side of the face, because of implication of the sensory fibres above their decussation. Occasionally the tongue is paralyzed on the same side as the muscles of mastication (Figs. 14 and 15).

In a case of tubercle of the pons observed by Dr. C. S. Potts and myself, the symptoms were weakness of the muscles of the left side of the face, excepting the orbicularis palpebrarum; weakness of the muscles of mastication on the left side, and fibrillary tremors of these muscles; weakness of the left external rectus, with the loss of power of associated movement of the eyes to the left, but with preservation of the power of convergence; diminution of the appreciation of touch, pain, heat, and cold in the right upper and lower limbs, right side of the trunk and neck, occipital region and ear; loss of stereognostic perception and of the sense of position on the right side; diminution of the appreciation of touch, pain, heat, and cold on the left side of the face and head; slight loss of appreciation of heat and cold on the right side of the face; anæsthesia of the conjunctiva, mucous membrane of the nose, mouth, and tongue on the left side; deafness in the left ear; loss of taste in the left anterior half of the tongue, and ataxia of both lower limbs, especially of the right. The tumor was confined to the left half of the pons.

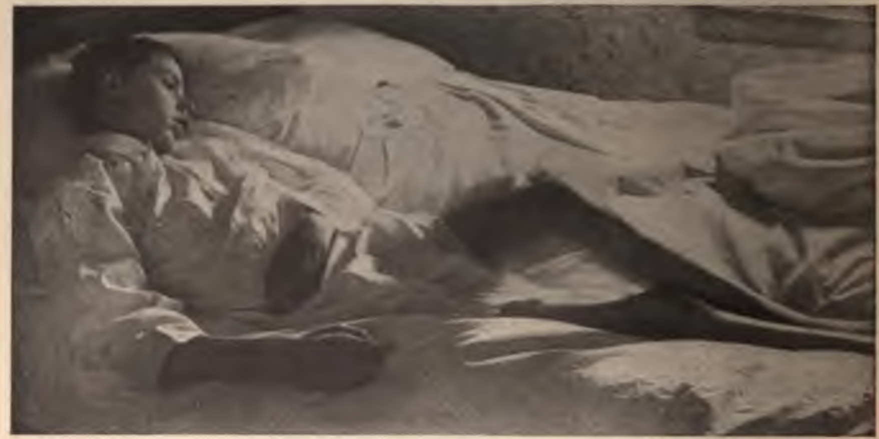

FIG. 18.—Photograph showing the tendency of the head to fall forward and to the left, and the presence of left hemiplegia in a case of glioma of the right side of the pons (Figs. 5-8).

I believe that this case shows that paralysis of lateral associated movement, when the power of convergence is preserved, is caused by a lesion of the posterior longitudinal bundle between the nuclus of the sixth nerve and that of the third. I have since the publication of this case studied two other cases of tumor within the pons with necropsy, in which the correct diagnosis could be made during the life of the patient. In these cases the symptoms were similar to those described above, and in each case I was able to observe paralysis of associated ocular movements toward the side of the tumor.

TUMORS OF THE CEREBELLUM.

Marcus Gunn[56] believes that intense double optic neuritis, with much swelling and surrounding retinal change, coming on quickly, sug-

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[56] Gunn, Brain, 1898, vol. xxi.

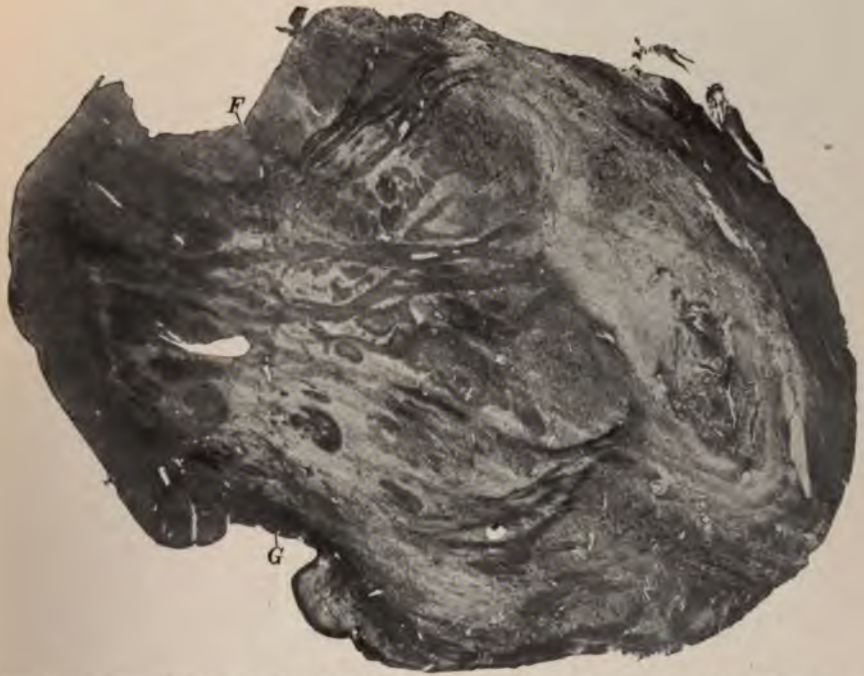

FIG. 19.—Glioma of the right side of the pons. F G. = the raphe of the pons. The right portion of the tumor is cystic. Same case represented in Fig. 18.

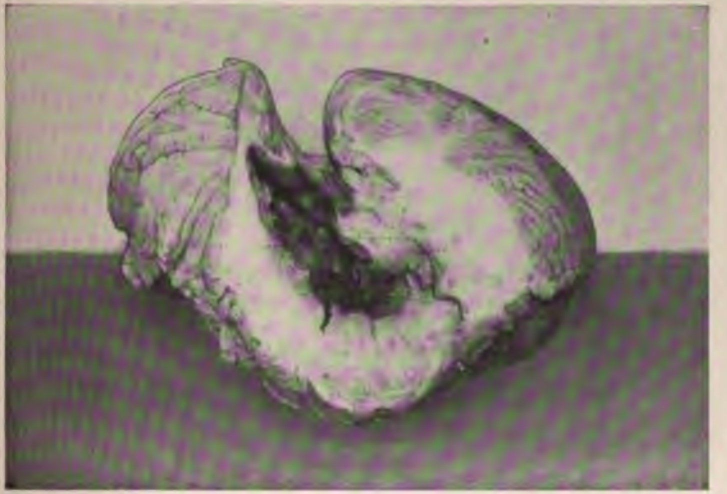

FIG. 20.—Cyst and glioma of the vermis.

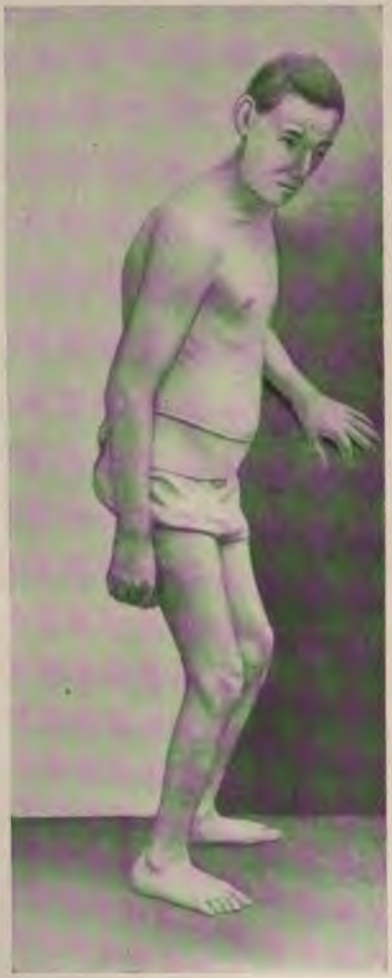

FIG. 21.—Position of standing in lesions of the cerebellum.

gests the cerebellum; and that one-sided optic neuritis, or marked difference in the intensity of the neuritis on the two sides, suggests the cerebrum, and is, on the whole, in favor of the tumor being on the same side as the excess of neuritis, where there are other reasons for localizing a tumor in the front of the cerebrum.

Cerebellar tumor, he says, very often excites a peculiarly intense optic neuritis, with great engorgement of the papillæ and marked œdema of the surrounding retina, with, not uncommonly, a macular stellate figure similar to what is seen in so-called albuminuric retinitis. This condition is seen also in basal meningitis, and possibly the reason for its occurrence with cerebellar tumor is that the latter is likely to cause basal meningitis. Mr. Gunn says he has found these changes in the eye-grounds in at least two cases of frontal tumor, in neither of which was there any evidence of basal meningitis.

The changes resembling those of albuminuric retinitis were present in a number of cases of tumor that have come under my observation.

Tumor of the cerebellum is often in connection with a cyst (Fig. 20), but the tumor may be so small that it may be overlooked, unless careful microscopical study is made; indeed, some writers believe that a cyst of the cerebellum is always at some time in its growth in connection with a tumor.

The ataxia of cerebellar tumor is often intense, and the patient staggers from side to side like a drunken person (Fig. 21). Choked disks are generally pronounced. The headache may be occipital or frontal, and in some cases may be associated with rigidity of the neck. A tendency for the head and trunk to incline forward and to one side (Fig. 18) is pronounced in some cases, but it is uncertain whether the inclination of head and trunk is toward the side of the tumor or away from it. Hemiasynergy is an unreliable sign, although I have seen it in three or four cases. It may be tested by having the patient while lying down draw up each lower limb separately, and it should be observed then whether the leg is extended on the thigh and the thigh on the trunk synchronously, or whether there is a dissociation of these movements. The latter is supposed to indicate the presence of a lesion in the lateral lobe of the cerebellum on the side of the hemiasynergy.

Vertigo is often an early and persistent sign of tumor of the cerebellum.

Staggering in walking, like the inclination of the head and trunk, may be toward or away from the side of the tumor.

Deafness, such as may be caused by involvement of the acoustic nerve may be a valuable sign pointing to the side of the cerebellum affected, and is likely to be present when the tumor is in the cerebellopontile angle.

In a case of cerebellar abscess I had the opportunity to observe that nystagmus was greater when the eyes were turned toward the side of the lesion than when they were turned away from it. I have not been able to make this observation in any other case, although the sign has been seen by others.

Tenderness to pressure over one side of the occipital region, and increase or diminution of vertigo according as the patient lies on one or the other side of the body, are signs of uncertain value.

The patellar reflex is frequently lost in cases of cerebellar tumor, but it may be exaggerated, or may vary in intensity from time to time.

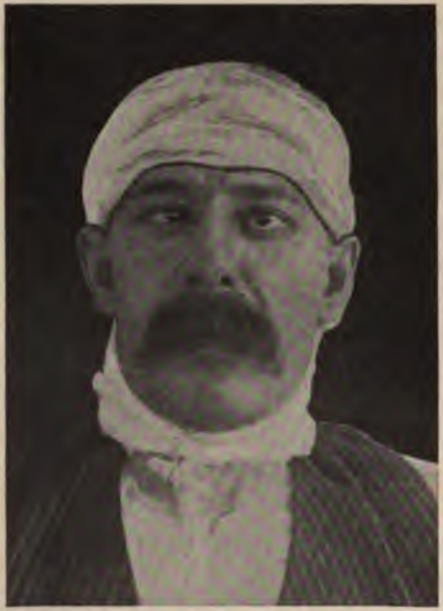

FIG. 22—Paralysis of each external rectus from a tumor in the fourth ventricle, implicating the abducens nuclei.

Lesions Simulating Tumor.—Other lesions of the brain may cause symptoms resembling very closely those of tumor, and chief among these is abscess. In most cases some purulent focus may be detected as the cause of the cerebral abscess, and most frequently it is disease of the middle ear. In other cases pus in the ethmoid or sphenoidal sinuses may be the starting point for cerebral abscess, or there may have been some injury to the bones of the skull. The symptoms of abscess are likely to develop more rapidly than do those of tumor and may be associated with fever, or subnormal temperature, chills, slowing of the pulse; or the symptoms of meningitis may be added,—viz., transitory ocular palsies, rigidity of the neck, Kernig's sign, etc. Purulent meningitis often accompanies cerebral abscess.

When the meningitis is in plaques a differential diagnosis from brain tumor may be impossible. This form of meningitis, described especially by the French writers, is more common in the motor area, and I have described one of the two cases reported in this country. In my patient the symptoms were precisely those of tumor of the parietal lobe.

Hydrocephalus in often associated with tumor and I have seen it so mask the symptoms of a tumor on the pons that the latter could not be diagnosticated. Again, I have seen internal hydrocephalus without tumor cause the symptoms of a cerebellar neoplasm.

Hemorrhage into the brain can be easily distinguished. The rapidity of onset and the course of the symptoms are unlike what is seen with brain tumor, but sometimes hemorrhage into a tumor occurs, and the diagnosis then is made chiefly by the sudden increase of symptoms.

The differential diagnosis between general paralysis of the insane (chronic meningo-encephalitis) and cerebral tumor usually is easy, but I have known the former to be regarded as tumor, and the danger of mistake is greater when the encephalitis is of rather rapid onset. Usually, however, a correct diagnosis can be made.

Aneurysms may give the symptoms of brain tumor. In some cases the stethoscope applied to the head may be of assistance, but I have known a supposed tumor at the chiasm causing bitemporal hemianopsia to be in reality an aneurysm in this region, and to present no signs characteristic of aneurysm.

CHAPTER X.

BULBAR AND PSEUDOBULBAR DISEASES

By E. W. TAYLOR, A.M., M.D.

The clinical significance of affections of the oblongata lies essentially in the fact that in this portion of the brain are gathered, in small compass, nerve mechanisms of vital importance. The hypoglossal nerve, and the group made up by the spinal accessory, vagus and glossopharyngeal, are situated here, in close relationship to each other. Disease processes, small in extent, may therefore lead to most serious disorders of function, if localized in the oblongata, and particularly in its dorsal portion. Owing to the great functional importance of this region, therefore, a separate consideration of its diseases is justified, although it should be borne in mind that a considerable number of the bulbar affections are simply local expressions of more general processes, either spinal or cerebral, or involving higher levels of the brain stem. For example, bulbar lesions are often associated with tabes, poliomyelitis, progressive muscular atrophy, including amyotrophic lateral sclerosis, multiple sclerosis, or with so-called Landry's paralysis, diseases which are ordinarily included in the spinal category. On the other hand, bulbar symptoms are not infrequently combined with generalized cerebral lesions, of which arteriosclerosis, paralytic dementia, and syphilis will serve as examples. More important for our present purpose is the association with encephalitis, and especially with superior poliencephalitis, ophthalmoplegia, and other affections of cranial nerve nuclei beyond the immediate limits of the oblongata.

As knowledge of pathological processes grows, it becomes increasingly evident that degenerative or inflammatory conditions are rarely sharply limited to one small area in the nervous system, although their clinical manifestations may point predominantly toward such localized involvement. This is somewhat strikingly true of the bulbar diseases, which, as already suggested, in many cases constitute merely the most conspicuous part of the clinical picture, owing to the highly important functional significance of this portion of the brain. It is still convenient, however, to use the accepted nomenclature and to describe the bulbar lesions under a separate heading.

General Symptomatology.—The symptoms arising from disease of the oblongata alone are chiefly dependent upon lesions involving the nuclei of the ninth, tenth, eleventh, and twelfth nerves. Through involvement of the vagus breathing is interfered with, leading to varying degrees of dyspnœa and at times to the Cheyne-Stokes type. The normal action of the heart may be disturbed, with irregularity of the pulse, or with an increased rapidity or abnormal slowness, depending upon the condition of paralysis or irritation of the heart regulatory mechanism. A more important and characteristic sign of bulbar injury is difficulty in deglutition, brought about by involvement of the accessory-vagus-glossopharyngeal group of nerves. Mastication may also suffer through disordered innervation of the hypoglossal nerve, and of the motor branches of the trigeminal to the jaw muscles. This latter difficulty is usually not conspicuous, since the degenerations in the motor nuclei of the fifth nerve lying in the pons are usually slight, if not entirely lacking. The part taken by the tongue in the act of mastication is, however, sufficient in all well-marked cases to render imperfect the disposal of food in the mouth. Paralysis and atrophy of the tongue is one of the most constant disturbances in the course of bulbar disease. This leads to difficulty in forming a bolus of food, and in passing it backward, and also to a characteristic speech disturbance which leads very early to difficulties in articulation, progressing from an imperfect enunciation of certain sounds (dysarthria) to an ultimate complete disability to articulate (anarthria). This is due not only to the paralysis of the tongue, but also to disturbances in the innervation of cheeks, lips, and palate, which determine the shape of the mouth cavity. Owing to difficulty of deglutition, as well as to probable disturbance in the centre in the oblongata, presiding over secretion, the saliva is apparently secreted in increased amount, and constantly flows from the imperfectly closed mouth. This combination of symptoms, more or less developed in various cases, constitutes the essential picture of the primary lesions of the oblongata.

A fairly sharp distinction may, in general, be made between those more chronic lesions which are sharply limited to the oblongata, and others of more acute onset and more extensive distribution. In the latter the general symptomatology becomes much wider in range, owing to the fact that the lesions are no longer limited to the bulb, but extend to the pons, and even in certain cases to the mid-brain region of the corpora quadrigemina and crura cerebri. This widening of the scope of so-called bulbar diseases is not strictly accurate, but is justified and necessitated by the diffuse character of many of the underlying lesions, and on the clinical side by the frequent uncertainty of accurate focal diagnosis of disease involving the pons-oblongata region. In general, the symptoms are dependent upon lesions of the nerves already mentioned, having their nuclei in the oblongata, and also upon the seventh, sixth, and fifth nerves located in the pons, together with the fourth and third nerves, lying in the quadrigeminal region. Paralysis of the face, disturbance in the innervation of the jaw, and various paralyses of the ocular muscles, are therefore frequent. Added to these are disorders of motility of the extremities from involvement of the ventrally lying pyramidal tracts, as well as varying alterations of sensibility, dependent upon interruption of sensory tracts. Except for the symptoms referable to lesions at a distance or of unknown cause, which will receive detailed discussion later, the symptomatology of this complicated region is wholly dependent upon a disturbance of function, either through irritation or destruction of its component parts, or through the cutting off of its central connections.

A distinction may in general be made between those affections which are degenerative in character and chronic in course, and those of acute, or sub-acute onset, in which the nerve elements are involved secondarily to vascular changes. Chronic progressive bulbar paralysis is a type of the first class, and so-called apoplectiform bulbar paralysis, brought about by embolism or thrombosis of arteries, a type of the second. A third general group includes those conditions in which the lesions apparently producing bulbar symptoms lie at a distance from the oblongata,—pseudobulbar palsy,—and finally those cases in which no lesions have been found sufficient to account for symptoms predominantly of bulbar type,—bulbar paralysis without anatomical basis; myasthenia gravis in some of its forms.

The following discussion will elucidate these more or less distinct processes, all of which have been differentiated within the past half century.

PROGRESSIVE BULBAR PALSY.

Terminology.—Various terms, suggestive of the location and of the course of this disease, have been used since the earliest description by Duchenne. The more important of these are: Progressive muscular paralysis of the tongue, palate, and lips (Duchenne); chronic progressive bulbar paralysis (Wachsmuth); progressive nuclear bulbar paralysis (Kussmaul); labio-glosso-laryngeal paralysis (Trousseau); progressive amyotrophic bulbar paralysis (Leyden).

History.—Following an observation of Dumesnil in 1859, in which the sug-·ion was made that bulbar lesions and progressive muscular atrophy might co-exist, Duchenne[1] in 1860–61 gave the first satisfactory description of bulbar paralysis. He critcised Dumesnil's observation on the ground that progressive bulbar paralysis and progressive (spinal) muscular atrophy were absolutely distinct diseases, the first being characterized by paralysis without atrophy, and the second by atrophy without paralysis, a false assumption, as later demonstrated. Observations by Romberg and Trousseau, previous to Duchenne's description, did not indicate a close conception of the character or manifestations of the disease. In 1868 Trousseau, on the basis of cases with somewhat unsatisfactory autopsies, completed Duchenne's earlier description, but maintained the essential identity of the disease with progressive muscular atrophy. Microscopic examination revealed certain degenerations of the hypoglossal and accessory nerves, but threw no definite light on the changes in the nuclei. The investigations of Baerwinkel, Wachsmuth, and Schultz definitely increased knowledge of the subject in the years up to 1865. Baerwinkel was struck with the apparent retention of electrical reactions in affected muscles, and also with the sharp localization to bulbar nerves. Wachsmuth in 1864 described certain alterations in the oblongata, which were, in part, correctly brought into relation with the clinical symptoms. The pathological anatomy was placed on a firm basis by the work of Charcot and Joffroy in 1869, verified and carried further by Charcot's pupils, as well as by Leyden (1870). The degeneration of certain motor nuclei in the oblongata, with the associated and resultant alterations of nerves and muscles, was determined as the essential lesion. Kussmaul (1871) urged the neuropathic character of the degeneration and established the analogy to progressive (spinal) muscular atrophy on a firmer basis. Cases thereafter were rapidly reported, with increasingly accurate descriptions, both on the clinical and anatomical side, by Gombault, Duchenne, Joffroy, Hun, Dejerine, Freund, Richie, Reinhold, Hoffman, Tooth and Turner, Remak, Turner and Bullock, and many others. At the present time the affection and its relations are adequately understood. The work of the past fifteen or twenty years has been to differentiate from the chronic progressive type of bulbar paralysis other varieties of bulbar disease which will be considered in detail later in this chapter.

Symptomatology.—Bulbar paralysis of the progressive type, both because it was first described and also because it presents a most characteristic picture of bulbar disease in general, may serve as a type of lesions of this region.

The symptoms, as observed in a typical case, are as follows: The onset is gradual, usually indicated by a slight disturbance of speech, to which small importance is at first attached. A persistence of this symptom, combined with a sensation of pressure about the neck, finally leads the patient to a realization of the abnormal condition. As in many forms of disease of insidious onset, a slight exacerbation of symptoms may give rise to the idea of a sudden onset, which in this affection may be assumed never actually to occur. With the progress of the disease the tongue is affected; its movements are hindered; subjective sensations of weight or stretching in the tongue and throat are noticed, which may extend downward. In conjunction with these symptoms the speech becomes more difficult, more indistinct and slower, and accompanied by feelings of fatigue. The indistinctness of utterance is brought about first by difficulty in the enunciation of certain letters, in general, inversely to the process of learning to speak. The sounds " r" and " sch" are early lost, as in the child they are last to be acquired. Later, as the movements of the tongue become more and more hindered, enunciation of the lingual consonants " l, r, n, and t," and afterwards " s," is lost (Gowers, Leyden, and Goldscheider, quoting Kussmaul, find that " s" is early lost); " t and d" are involved when the tongue can no longer be brought firmly against the palate. Succeeding or associated with the palsy of the tongue, the lips are involved, with the result that speech is still further hindered in the pronunciation of the labials, " o, u, p, b, m." Even when the disorder has reached this stage words may still be correctly enunciated by special effort and deliberation. The vowel sound " a" is longest retained. Finally, with the advent of weakness of the palate, complete paralysis of the tongue and inability to use the lips, speech is reduced to a few meaningless sounds, which bear only a distant similarity to articulate language.

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[1] Duchenne: De l'électrisation localizée, 2d ed., p. 621, 1861; also Arch. Gén. de Méd., xvi. 283, 1860.

Before this final stage is reached, difficulties in deglutition supervene, due to the combined weakness of the tongue, soft palate and pharyngeal muscles. Food can no longer be properly moved into the back of the mouth cavity by the tongue, and the act of swallowing is likewise difficult. Mastication also suffers from the palsy of the tongue. Liquids are regurgitated through the nose, and with the progressive weakness of the muscles of the epiglottis particles of solid food enter the larynx. Swallowing becomes increasingly difficult, and semisolids are found to be more readily taken than either solids or liquids, owing to the lesser likelihood of finding lodgement in the larynx or being regurgitated through the nose. Along with the difficulty in swallowing, saliva collects in quantity, and finally runs in a constant stream from the mouth, which offers small resistance on account of the palsy of the lips. The patient at this stage presents the characteristic picture of bulbar disease, practically speechless, swallowing with the utmost difficulty, and continually wiping away saliva as it flows from the imperfectly closed lips.

As the disease still further advances, the muscles governing the vocal cords are apt to suffer; paralysis of the cords results and phonation finally becomes impossible. Coughing ultimately becomes difficult, with the accompanying danger of inhalation pneumonia. With such a gradual increase of paralysis in tongue, lips, pharynx, and finally larynx, the disease runs its course. As Gowers puts it, " the symptoms are grouped about the tongue as a centre," but they extend far beyond the its of this one organ, before life is actually threatened.

Other symptoms which occur, but do not dominate the typical clinical picture, should be mentioned. Although there is no loss of sensibility, and taste is usually unaffected, the throat reflexes ordinarily fail, owing to the motor involvement, though this is not invariably the case. This tendency to loss of reflex activity naturally renders deglutition more difficult and increases the danger of the entrance of food into the larynx. The jaw reflexes, on the contrary, may be increased, no doubt due to a degeneration of the pyramidal tracts. (See Pathological Anatomy.) Dyspnœa, rapid pulse, œdema of the lungs, have been attributed to late involvement of the motor vagus (accessory). Except for an undue emotionalism, laughter or tears from insufficient cause, the mental faculties remain unimpaired, and the patient is painfully conscious of his sufferings up to the very end of the disease.

Physical Signs.—The physical signs of the affection are as unmistakable and characteristic as its symptomatic course. The nerves involved are the hypoglossus, the motor portions of the vagus-glossopharyngeal-accessory group, rarely the motor portion of the trigeminus, the facial, and ocular nerves. The involvement of these latter nerves is rather to be regarded as a complication than as a component part of the symptom-complex, assuming, as we do somewhat arbitrarily, that progressive bulbar paralysis is a disease *sui generis,* limited to the oblongata. As a direct result of the primary degeneration of the hypoglossal nerve, the tongue atrophies; as the disease progresses, it becomes wrinkled, and furrowed, often swollen in appearance, and lies practically immovable on the floor of the mouth. A similar atrophy occurs in the lips; a complete closing of the mouth finally becomes impossible, and the lower portion of the face assumes a characteristic expressionless appearance in marked contrast to the usually alert and distressed aspect of the eyes and upper portion of the face.

The fact that the upper distribution of the seventh nerve is spared, whereas the lower branches are apparently involved in the degenerative process, has led to considerable speculation as to the course of the lower fibres, with the general conclusion, as just stated, that the orbicularis oris is supplied either directly or indirectly by fibres of the hypoglossal nerve, but connected with the facial through the dorsal longitudinal fasciculus. The close physiological association of lips and tongue in phonation renders probable the assumption that the innervation is from a common source, or from areas in intimate anatomical relationship. It is, at least, certain that atrophy of the lip muscles is a constant feature of progressive bulbar palsy, whereas, in exceptional instances only are the upper facial muscles involved. In certain cases of bulbar paralysis, however, forming part of a general amyotrophic lateral sclerosis, the facial nerves may be involved in all branches; in other cases, as reported by Remak,[2] Tooth and Turner,[3] Turner and Bullock,[4] and others, the evidence goes to show that in spite of nuclear and other degeneration of the seventh nerves, the upper branches have still been spared. Following an earlier suggestion of Mendel,[5] that the upper facial muscles were supplied by fibres of the third nerve, through the dorsal longitudinal fasciculus and the trunk of the facial nerve, Tooth and Turner were able to demonstrate a complete atrophy of the nucleus of the seventh nerve and also of its dorsally directed fibres and the knee, whereas the outgoing nerve contained normal fibres. From this observation, and others of similar character, it has been assumed that the upper facial muscles derive their supply indirectly from the nucleus of the third nerve. This would explain the sparing of the upper branches in those cases of bulbar palsy in which the lower branches of the seventh nerve were manifestly involved without assuming the intervention of the hypoglossal. It would also fall in with the physiological hypothesis, as already suggested, that muscles acting in association have a closely

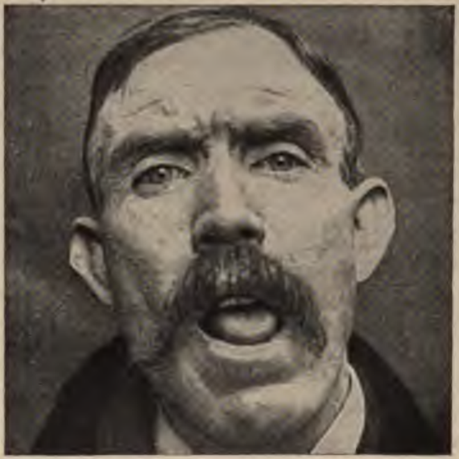

FIG. 1.—Progressive bulbar paralysis, patient of 39, six months duration. Deglutition very difficult; speech wholly impossible; palsy of tongue, lips, and muscles of deglutition; exaggerated deep reflexes The photograph shows an extreme attempt to open the mouth and protrude the tongue.

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[2] Remak: Arch. f. Psych., xxiii., H. 3, 919, 1892; Neurol. Ctb., vii. 62, 1888; idem, x. 28, 1891; idem, xi. 753, 1892; Berl. klin. Woch., xxxii. 29, 1895.

[3] Tooth and Turner: Brain, xiv. 473, 1891.

[4] Turner and Bullock: Brain, xvii. 693, 1894.

[5] Mendel: Berl. klin. Woch., xxiv. 913, 1887; Neurol. Ctb., vi. 537, 1887.

related nerve supply,—lips and tongue in articulation, opening and closing of the lids through the action of the levator palpebræ (third nerve) and the orbicularis palpebrarum (seventh nerve, through fibres derived from the third nucleus).

On the other hand, Wilbrand and Saenger[6] are of the opinion that the nucleus of the third nerve has nothing whatever to do with muscles supplied by the upper facial. In support of this opinion they quote a careful examination by Cassirer and Schiff in which there was no disturbance in the function of the upper facial in spite of complete degeneration of the oculomotor nucleus, including its dorsal portion, to which Mendel and others had attached particular significance. Siemerling and Boedecker also oppose the idea that the facial distribution to the eye muscles has any relation whatever with the nucleus of the third nerve. Evidence is, therefore, as yet conflicting as to the course of the fibres innervating the upper portion of the face, although the fact must be accepted that in nearly all cases of chronic bulbar paralysis the lower facial muscles are alone involved. The possibility of at least a partial nerve supply from the region of the hypoglossal nucleus for these muscles must be considered, as already suggested.

In those cases of otherwise uncomplicated bulbar paralysis, in which the upper branches of the seventh nerve have been involved in the paralysis, it is probable that we have to do with a family type of the disease, as observed by Londe[7] and Remak.[8] Londe found in a child bulbar paralysis, with general involvement of the upper branches of the facial nerve, together with palsy of the levator palpebræ superioris, but without other ophthalmoplegia. Remak has also reported a number of cases of involvement of the upper facial muscles in children, which led to the suggestion of a special type of family bulbar paralysis, having analogies to, but not identical with, the adult form.

In general, regarding the greater or less involvement of the upper cranial nerves, lying in the pons or above, and including the facial nerve, it is now sufficiently evident that the underlying process is by no means a sharply limited one, but that, as already suggested, it may extend both upward and downward from its point of earliest development in the nuclei of the bulb proper. This fact will receive further comment in the discussion of the pathological anatomy.

Electrical Reactions.—The electrical alterations in bulbar paralysis, as in other analogous affections, depend upon degenerative changes in the peripheral motor neurones with consequent muscular atrophy. The early confusion inaugurated by Duchenne, and later set at rest by Trousseau, Kussmaul and all later observers regarding atrophy and paralysis, and the relations of bulbar palsy to progressive muscular atrophy, was enhanced by the apparent failure of electrical changes in the tongue. It is, however, undoubtedly true that if the muscles of the tongue could be stimulated with the same ease as other muscles, electrical alterations of greater or less degree would always be found. The deposition of fat in the atrophying tongue is one of the chief hindrances. Observations hitherto made have not demonstrated a complete reaction of degeneration, but partial degenerative reaction (Erb), and various stages between that and the normal have been observed so frequently as to substantiate the *a priori* assumption of electrical alterations of some degree in all cases in which the hypoglossal nerve is degenerated. Paralysis of the tongue and neighboring parts from other lesions than those affecting the peripheral motor neurones naturally would not give rise to electrical changes, and no doubt it is these cases which have led to the confusion.

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[6] Wilbrand and Saenger: Die Neurologie des Auges, Bd. I., Abt. 1, p. 114.

[7] Londe: Rev. de Méd., xiii. 1020, 1893; xiv. 212, 1894.

[8] Remak: loc. cit.

Duchenne and later Dejerine[9] have drawn attention to the fact that the paralytic symptoms of bulbar paralysis may be explained by degeneration of the pyramidal tracts. To this may be associated degeneration of peripheral neurones, giving rise to a bulbar amyotrophic lateral sclerosis, or an amyotrophic lateral sclerosis of descending course. A paralytic and atrophic form have been distinguished. It is in general evident that the atrophic element, with altered electrical reactions, will predominate when the peripheral neurones are involved; that the spastic element will be conspicuous if the central neurones (pyramidal tracts) are involved, and that transitional forms may occur with varying degrees of degeneration of the two sets of neurones. The analogy is complete to spinal progressive muscular atrophy of the flaccid and spastic (amyotrophic lateral sclerosis) types respectively. Finally it should be remembered that progressive bulbar paralysis, if not too quickly fatal, represents either the starting of a degenerative process, which tends to progress beyond the confines of the bulb, or else develops as the final stage of analogous affections originating in the upper spinal cord.

Pathological Anatomy.—Progressive bulbar paralysis is characterized by a chronic, primary degeneration of the cells of the motor nuclei of the oblongata. This degeneration is not necessarily associated with primary alterations of the vessels, nor with evidences of inflammatory reaction, and should be distinguished from those affections of the oblongata which are dependent upon vascular disorders or inflammatory processes. (See following sections.) The cells of the hypoglossal nuclei suffer most extensively; many of them show marked degenerative changes, are shrunken or entirely disappear. The accessory hypoglossal nuclei are spared. Less marked, but still evident, are degenerations in the motor nuclei of the accessory nerve and of the cells of origin of the motor vago-glossopharyngeal roots. Degenerations have at times been found in the nucleus ambiguus, also in the nuclei of the seventh pair and motor nucleus of the fifth nerve in those not infrequent cases in which the disease progresses beyond the oblongata into the pons. Unless we regard the dorsal nucleus of the vagus and glossopharyngeus as motor, at least in part,[10] it is difficult to account for the paralyses of larynx and pharynx, in those cases in which the nucleus ambiguus has been found intact, unless their supply be wholly through the accessory nerve. These matters require further investigation on the pathological-anatomical side.

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[9] Dejerine: Arch. de Physiol., xv. 180, 1883.

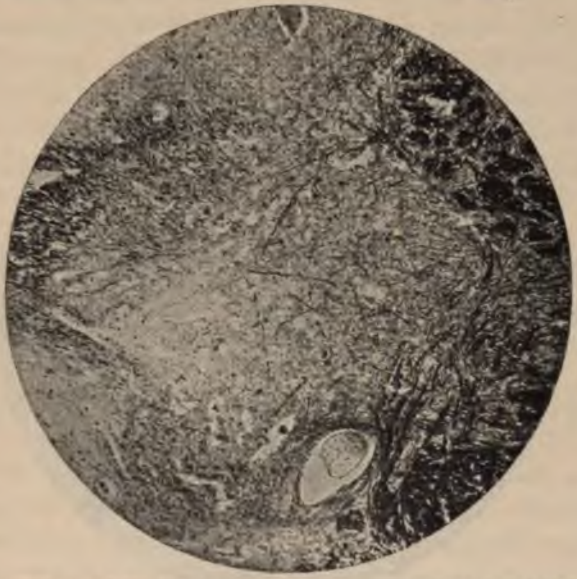

FIG. 2.—Progressive bulbar paralysis; nucleus of hypoglossal nerve in centre of field, showing loss of myelinated fibres and almost complete disappearance of nerve cell bodies; x 45. (Photograph by W. S. Greene.)

Corresponding to these cell degenerations the hypoglossal network of myelinated fibres is degenerated, and the intra- and extra-bulbar axones of the affected cells. The ensuing muscular atrophy shows no peculiarity. The muscle-bundles, particularly of the tongue, are thinned, the connective tissue increased, granular and fatty degeneration occur often with an excessive deposit of fat, which may somewhat mask the atrophy. The tip of the tongue suffers most.

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[10] Spiller: Univ. of Penna. Med. Bulletin, March, 1903.

A very frequent, though not absolutely constant, accompaniment of the muscular alterations is degeneration of the pyramidal tracts. Opinions differ as to the frequency of this association. A number of cases have been described in which no alterations in the pyramidal tracts were found; they may, nevertheless, have been present in very slight degree, as might have been shown had the Marchi method been used. It has been suggested (Remak) that rapidly fatal cases are less likely to show degeneration of the pyramidal tracts, but that such degeneration would probably have developed had life been prolonged. This question is precisely analogous to that long discussed with regard to progressive muscular atrophy with or without degeneration of the pyramidal tracts. In general, the evidence goes to show that the peripheral neurones may be degenerated independently of the central, but that the converse very rarely occurs.

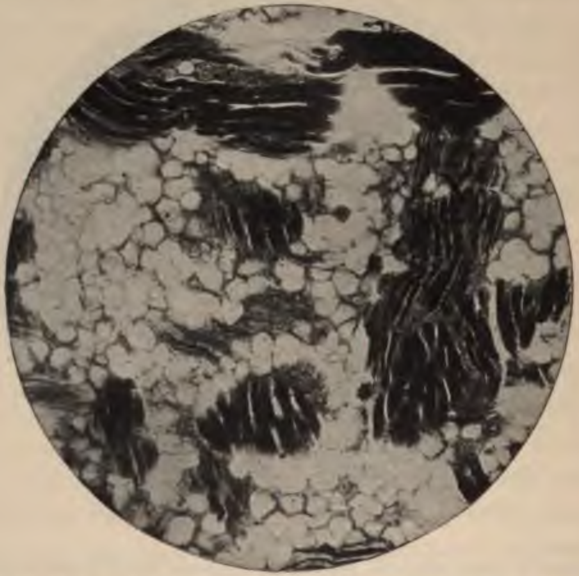

FIG. 3.—Progressive bulbar paralysis; tongue; atrophy of muscle bundles; increase of fat; x 45. (Photograph by W. S. Greene.)

Etiology, Course, and Treatment.—The cause of the affection is wholly unknown. Syphilis and alcohol have no direct bearing on its development. It occurs more frequently among men than women—34 men and 19 women in a series of 53, as observed by Kussmaul. Apart from the rare, apparently congenital type, observed in children, it is distinctly a disorder of the latter years of life, most frequent between the fiftieth and the seventieth years. I have, however, recently observed two cases in men, each thirty-nine years old.

The course is from one to three years, and in certain cases much longer. In general, it is steadily progressive and death results from paralysis of respiration and deglutition, or from inhalation pneumonia in consequence of pharyngeal paralysis. The prognosis is, therefore, wholly bad, although occasional remissions may occur.

The treatment must be directed towards proper diet, especially in the later stages. Semisolid food is usually indicated, and when swallowing becomes too difficult or impossible, the stomach tube becomes a necessity. General hygienic measures are essential, and encouragement serves better than drugs. Strychnia in increasing doses may be used; its efficiency is doubtful. Kussmaul and later Remak have urged the value of the galvanic current for the purpose of stimulating the muscles concerned in swallowing. Remak advises that the anode be placed at the back of the neck, and the cathode passed over the neck on either side of the trachea, using a current of 3–6 milliamperes.

ACUTE (APOPLECTIFORM) BULBAR PARALYSIS.

Hemorrhage, Embolism, Thrombosis of Arteries of Bulb and Pons.—Under this general heading may be included those affections of the oblongata and pons, which are characterized by relatively sudden onset, and are either rapidly fatal or else show a tendency towards improvement. They are to be distinguished from the foregoing progressive bulbar paralysis on the clinical side by their non-progressive course, and on the anatomical side by their wholly different pathological alterations.[11]

Inasmuch as the lesions producing the symptoms of acute bulbar paralysis are mainly dependent upon the blood supply of the oblongata-pons region, the following anatomical facts should be borne in mind: The two vertebral arteries unite to form the basilar artery at the lower border of the pons, having given off, among others which do not now concern us, branches to the oblongata. The basilar artery before dividing into the posterior cerebral arteries gives off transverse branches to the pons. These arteries run for a distance on the surface of the pons and then penetrate its substance. From studies made by Duret, it appears that the nuclei of the hypoglossal and accessory nerves receive their blood supply from the anterior spinal artery and the vertebrals; the nuclei of the vagus, glossopharyngeal and auditory from branches from the upper portion of the vertebrals, or lower part of the basilar; the facial, trigeminal, and ocular nerves from the branches of the basilar. Clinical experience, however, has not as yet justified complete reliance on these facts.

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[11] Van Voort (Deuts. Ztschft. f. Nervenheilk., viii. 137, 1896) has published a valuable discussion, with an elaborate table, on Acute Bulbar Paralysis, and very recently Dana (Trans. Ass'n American Physicians, xviii. 446, 1903) has published an excellent and comprehensive paper on the same subject.

Pathological Anatomy.—The lesions underlying a large proportion of the acute bulbar affections are alterations in the blood-vessels, which lead secondarily through weakness of their walls to hemorrhage, or through roughening or narrowing of the lumen to thrombosis, and when the proper conditions are present, to embolism. The sources of the vessel changes, and their consequent failure to carry on their normal function, are those recognized in arterial disease generally,—cardio-renal affections, syphilis, senility and the arteriosclerosis which is common in middle life and almost physiological in old age. Emboli may also reach this region from diseased heart valves. The basilar artery is a frequent seat of marked sclerotic change, and often shows a

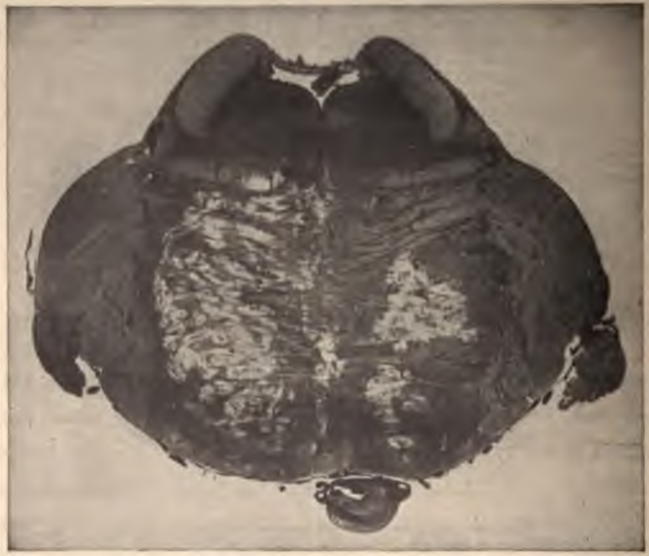


FIG. 4.—Acute (apoplectiform) bulbar paralysis; pons: lighter areas softening, as result of thrombosis of basilar artery. (Photograph by W. S. Greene.)

tortuous course, irregularly thickened walls, and aneurismal dilatations which may exert destructive pressure on the overlying oblongata. The same is true, though perhaps to less degree, of the vertebrals. The pons and oblongata must, therefore, both suffer from such lesions, since their immediate blood supply is wholly from these larger trunks. The softenings, or more rarely, hemorrhages, which may result, do not differ histologically from such lesions observed elsewhere in the brain, and require no detailed description here. The character of the blood supply naturally gives rise to the possibility of multiple lesions. The pons and oblongata, for example, are often simultaneously involved, and it is frequently impossible to localize the lesion accurately in one or the other region, hence the general inclusion of the symptoms under the head of bulbar paralysis. A higher level than the pons may also at times be involved, still further extending the inclusiveness of the term. Owing to the facts that the lesions, particularly of softening, may be multiple, that they are often extensive, and that they show no special predilection for the gray matter, it follows that the symptoms produced are far less definite than in the circumscribed chronic progressive form, and therefore that wide-spread disorders of motility and sensibility may result.

BULBO-PONTINE HEMORRHAGE—ACUTE BULBAR APOPLEXY.

Symptomatology.—Hemorrhage of the pons is rare; it has been estimated that hemorrhage of the brain (cerebrum) is about fifty times as frequent. Hemorrhage of the oblongata is still less common. It occurs most frequently in the fourth decade of life, and is usually due, as in the brain, to the results of cardiac hypertrophy in the course of renal degeneration associated with arteriosclerosis. The branches of the basilar are the usual sites of rupture; the basilar itself is rarely involved.

The symptoms in a typically severe and fatal case, following in general Dana's description, are: Prodromal neck pain, headache, vertigo, vomiting, succeeded at varying intervals by an apoplectic seizure often of extreme suddenness. This is usually followed by profound coma, twitching of face and extremities, but rarely a general convulsion. The face is flushed; the temperature subnormal at first; pulse irregular; respiration slow, irregular and of the Cheyne-Stokes type. The pupils are contracted; there may be convergent strabismus or conjugate deviation. Later a rise of temperature occurs, often reaching a high point; swallowing is impossible; dyspnœa, rapid pulse, cyanosis, due to paralysis of respiration, lead to death in from six to twenty hours after the onset. In less violent cases paraplegia or a crossed paralysis manifests itself when the acute stage is past, with exaggeration of reflexes.

BULBO-PONTINE SOFTENING.

The clinical differentiation between hemorrhage and softening is usually difficult and often impossible to make, as in the case of similar lesions in the cerebral hemispheres. The suddenness of onset, the presence of conditions in other organs predisposing to rupture of blood-vessels, notably hypertrophied heart with elevated blood-pressure, are suggestive of hemorrhage, though never absolutely conclusive. Softening in the pons and oblongata is about three times as frequent as hemorrhage, and occurs usually in syphilitic persons between the ages of thirty and fifty. Arteriosclerosis in some form is a necessary forerunner of softening, except in the rare cases of embolism. Men are affected as frequently as women.

In general, the onset of severe symptoms is slower in softening from any cause than in hemorrhage, with the possible exception of embolism. Following general signs of arterial disorders, such as headache, vertigo, tinnitus, marked bulbo-pontine symptoms develop with greater or less rapidity. If the oblongata be particularly involved, through disturbance in the branches of the vertebral arteries, disorders of speech and difficulty in deglutition occur, with possible paralysis of the pharyngeal and laryngeal musculature. Contraction of the pupil, with smaller palpebral fissure, retraction of the globe and facial flushing, may occur from involvement of a sympathetic centre supposed to lie in the oblongata. Further disorders of sensibility and motility supervene if the lesion be extensive, due to injury of the pyramidal tracts, and interruption of the sensory tracts lying deep in the oblongata. In unilateral lesions hemiplegia is a common accompaniment. There is also at times a dissociation of sensation of the usual character—loss of pain and temperature senses, with retention of the sense of touch, and ataxia. Defective sense of position, astereognosis, and forced movements have also been observed.

Involvement of the pons to the exclusion of the oblongata implies a freedom of the nuclei and peripheral distribution of the eighth to the twelfth nerves inclusive. The fifth, sixth, seventh, and often the third, show defects of various degree. Sensory disorder in the distribution of the fifth nerve is not uncommon. The pyramidal tracts, or often one tract, is involved, giving rise to hemiplegia, sparing the face if the facial nerve escapes injury both in its central and peripheral course. The so-called alternating or crossed hemiplegia is produced when the lesion lies in such a position as to involve the peripheral neurone of the seventh or sixth nerve, or if higher up, of the third nerve, and the pyramidal tract of the same side. The crossing of the pyramidal tract at a lower level leads to a paralysis of arm and leg on the opposite side, and of face or certain eye muscles on the same side as the lesion. Lesions involving the central path of the sixth nerve may also lead to conjugate deviation of the eyes towards or away from the lesion, depending upon whether the lesion be paralyzing or irritative. An injury to the common centre for the sixth nerve on one side, and the third nerve (branch to the internal rectus of the opposite side), if it be destructive, will lead to a conjugate deviation away from the lesion. In the absence of knowledge regarding the extent of injury, definite rules concerning the diagnostic focal value of conjugate deviation cannot be laid down. Convergent strabismus is a result of implication of the sixth nerve, one or both. Nystagmus also is a common sign of pons lesion, to be explained by disorder of the coördinative mechanism. Quadrigeminal lesions give rise to disorders in the distribution of the third nerve—external strabismus, ptosis, paralytic dilatation of the pupil—and of the less important fourth nerve. Symptoms of true bulbar paralysis, involvement of tongue, larynx, pharynx, may occur from interruption of the upper neurones of the hypoglossal, and vago-glossopharyngeal group in the pons. This effect is most markedly produced by a bilateral lesion. In such a lesion, the element of atrophy will naturally be lacking in the peripheral portion of the nerves involved. Injury of one fillet produces an imperfect sensory loss on the opposite side of the body, which may be associated with ataxia. Ataxia of the cerebellar type is probably produced by lesions of the superior peduncles and red nucleus. Pain is never a conspicuous symptom, which is in general true of intracerebral or spinal lesions. Uncontrolled emotionalism—forced laughter—is a frequent symptom in bulbo-pontine disease, and is perhaps best explained by the theory of partial or complete destruction of the paths of control, which must be closely associated if not identical with the upper neurones of the bulbar and pontine nerves.

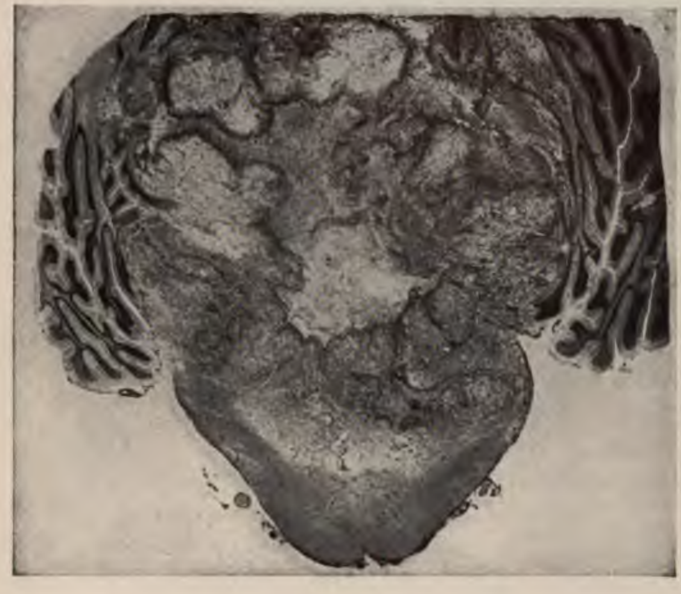
In general, the multiplicity of symptoms which may result from lesions of the oblongata or pons, or both, is apparent when one considers the complexity of anatomical structure in this portion of the brain. Considering, also, the fact that the ordinary lesions, producing acute bulbar paralysis, may vary widely in extent and destructiveness, it follows that the symptoms may range from slight and temporary disturbances in the functions of the bulb and pons up to conditions which are quickly fatal. It should, however, not be difficult to picture the symptomatology of intermediate stages, if one bears in mind the functional significance of this lower portion of the brain stem. The following case illustrates sudden bulbar palsy of slight degree:

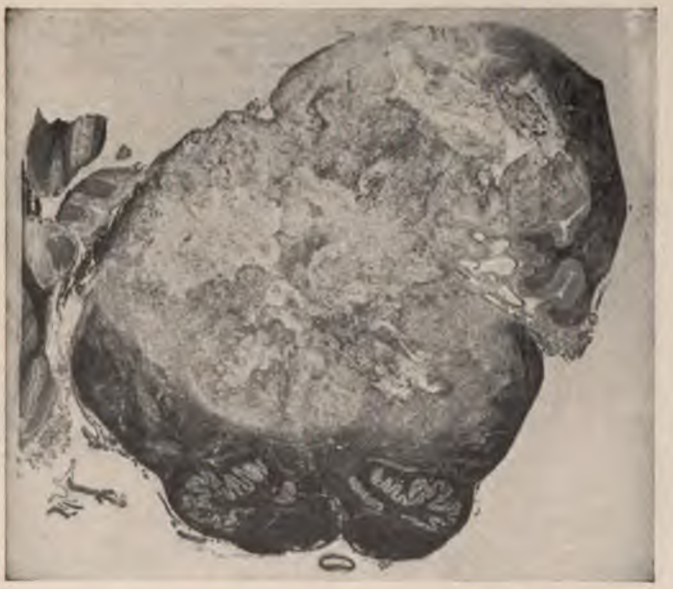
A woman of fifty-five, of active life, well except for phlebitis some years ago and an unexplained attack of vertigo several months before, suddenly without prodromata, noticed distinct difficulty in speech, coming on without pain. This was associated with difficulty in swallowing, but without actual regurgitation. Examination four days after the onset gave the following results: No definite arteriosclerosis: heart sounds normal, except for accentuated aortic second. The eyes showed nothing abnormal, and sensibility was, in general, unimpaired. The upper cranial nerves were not involved. The tongue was protruded imperfectly, and all movements were possible, though weak and slow. Speech was thick, laborious, but understandable. There was paresis of the soft palate and of the pharynx. Swallowing was very difficult, but possible, when either liquid or solid was taken in small amount. The reflexes were, in general, active, but there was no evidence of hemiplegia or other paralysis of the extremities. Later reports showed rapid improvement.

Such a case must be interpreted as due to a vascular bulbar disorder, not sufficiently extensive to lead to irremediable defect, It serves as a type of those bulbar affections which are slight in themselves, but are indicative of the possibility of more serious results in the future.

Course and Outcome.—As already suggested, if not immediately fatal, the tendency of this group of affections is towards improvement, usually leaving greater or less functional defect, except in the slightest cases, as for example, that quoted above. The reason for this is obvious from the character of the underlying pathological process, and is perfectly analogous to such processes elsewhere. Inasmuch as the conditions which lead to the onset of the symptoms are usually not materially influenced by treatment, the danger of subsequent attacks of similar character is evident.

Treatment.– The treatment of acute bulbar paralysis should primarily be directed to the underlying cause, hemorrhage or softening, and should be chiefly preventive. If syphilis be thought a possible cause, and it should be suspected, especially in relatively young patients, an anti-syphilitic cure should be administered vigorously. In other cases of arterial lesions, care should be taken to provide for adequate nutrition and to avoid the dangers of inhalation pnuemonia, as for the chronic progressive form. More important than the treatment of the actual condition are prophylactic measures, care of the kidneys and of the whole circulatory system. A strict dietary *régime*, avoidance of exertion and a restrained manner of life generally should be insisted upon in those cases which show the often vague signs of cerebral circulatory





FIGS. 5, 6.—Oblongata: gumma involving region of bulbar nerves, fourth ventricle and a part of the cerebellum; various somewhat irregular bulbar and general symptoms; basilar artery normal. (Photographs by L. S. Brown.)

disturbance, or which have undergone a slight apoplectic attack either of the brain or pons-oblongata region. Drugs are not serviceable, except to meet indications as they arise.

BULBAR PARALYSIS FROM OTHER CAUSES.

In addition to the foregoing somewhat definite type of bulbar disease, many other causes of disturbance in the function of this region have been described, some of which, on acount of their rarity and lack of relation to the general subject-matter of this book, may be cursorily considered. A few cases have been described (Leyden, Etter) of myelitis of the bulb, with symptoms of acute bulbar paralysis. Leyden describes and pictures a case in which inflammatory changes and multiple foci of hemorrhage were found in the region of the olives and elsewhere throughout the bulb, showing markedly on the floor of the ventricle. This condition is to be regarded as a local manifestation of the group of conditions usually described as acute encephalitis, or poliencephalitis, and corresponds in a measure to the condition of superior encephalitis, of which special mention is hereafter made (page 452). It serves to demonstrate the fact, not infrequently observed, that inflammatory processes, with a certain predilection for the gray matter, may occur anywhere throughout the central nervous system, the local symptoms naturally depending upon the part involved.

Trauma of the bulb occasionally occurs through stabbing wounds or fracture and dislocation of the upper vertebræ. Such injuries are usually fatal, but if not, the familiar symptoms are produced in proportion to the extent and severity of the lesions.

Bulbar symptoms in the course of typhoid fever and leukæmia have been described (Eisenlohr) and also in multiple neuritis. An infective agent (streptococci) was found in the fatal typhoid fever cases, hemorrhages and inflammatory changes occurred in the sheaths of bulbar nerves in the cases of leukæmia, and in rare cases it has been shown that a neuritis may affect the bulbar nerves.

Tumors in the posterior cranial fossa, meningeal thickenings from syphilis or other cause, aneurismal dilatations or actual aneurisms of the basilar artery, and more rarely of the vertebrals (Oppenheim, Seimerling) may by compression lead to bulbar symptoms, often with involvement of the pyramidal tracts, and consequent spasticity of the extremities (Figs. 5 and 6). The symptoms are naturally variable; they are usually slowly progressive, often with a final severe exacerbation. Owing to the tortuous course of the artery in compression by aneurism, the motor symptoms are irregular in distribution, and often of the type of an alternating hemiplegia. The principles of diagnosis apply in general to cases of this type as to others, a peculiarity being gradual course, with final increase of symptoms, either through softening or rupture of the aneurism.

Cases of a family, congenital type of bulbar paralysis have been described by Remak, Londe, and others, to which allusion has previously been made, in which the upper branches of the facial nerves are apt to be involved, a fact possibly of diagnostic significance.

SUPERIOR ENCEPHALITIS (WERNICKE).

On a basis of three cases Wernicke,[12] in 1881, described a new symptom-complex, to which he gave the name of acute hemorrhagic superior poliencephalitis. A previous case, reported by Gayet in 1875, had not served to establish the identity of the process. Wernicke regarded the affection as an independent, acute inflammatory process in the region of the nuclei of the ocular nerves, fatal in from ten to fourteen days. The focal symptoms consist in associated paralysis of ocular muscles, of quick onset and rapid progress, finally leading to a practically total paralysis of the muscles involved, with the possible exception of the sphincter iridis or the levator palpebrarum. The gait of the patient is ataxic, suggesting alcoholic intoxication. The mental state is markedly disturbed; somnolence and agitation may both occur; a mental condition strongly suggesting dilirium tremens may form part of the clinical picture. In the three cases described by Wernicke changes of an inflammatory character were observed in the optic disk. Alcohol played a prominent part in the etiology of the affection in two cases, and sulphuric acid poisoning in the other. The essential pathological alteration in each case was multiple punctiform hemorrhages in the dorsal portion of the third ventricle and the region of the aqueduct of Sylvius, including especially the nuclei of the ocular nerves.

Subsequent observations have extended the knowledge of the acute poliencephalitis described by Wernicke. Influenza is probably an important factor in etiology as well as other infectious diseases, and poisonings of varied character. The prognosis is also not so invariably bad as at first supposed, nor may the pathological anatomy be regarded as uniform.

In twenty well observed cases of Wernicke's hemorrhagic poliencephalitis, fifteen with autopsies, Wilbrand and Saenger[13] note the following facts of importance with reference to the ocular conditions: In nine cases there was ptosis of varying degree; in all the cases, excepting two, in which no observation on the condition of the muscles is made, the ocular muscles were variously involved, affecting both the third and the sixth nerves, giving rise to more or less immobility of the eyes, with accompanying diplegia, nystagmus, and strabismus; the pupils showed varying reactions to light, from normal to complete immobility; practically no observations were made on the power of accommodation, for obvious reasons, in view of the constant palsy of the external muscles; ophthalmoscopic examination showed changes in a few instances, of the character of hyperæmia, unusual pallor of parts of the disk, and occasionally papillitis. Various nuclear and peripheral degenerations were found post-mortem. In general, the ocular conditions showed no tendency to conform to any one type.

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[12] Wernicke: Lehrbuch der Gehirnkrankheiten, 1881, § 47, p. 229.

[13] Wilbrand and Saenger: loc. cit., p. 270.

The position definitely taken by Wernicke that superior poliencephalitis is a wholly analogous affection to poliomyelitis and other inflammatory affections throughout the brain stem has been amply borne out by subsequent observations. This is shown, first, by the essential identity of the pathological findings in these variously localized processes, and secondly, by the many transitional forms which have been described.[14] Encephalitis, on the one hand, superior poliencephalitis, inferior poliencephalitis, giving the symptoms of an acute bulbar paralysis, poliomyelitis, and finally poliencephalomyelitis, no doubt represent simply different expressions of a common underlying cause, the symptoms naturally varying widely with the portion of the nervous system involved.

The analogy may usefully be carried further in considering the more chronic forms of degeneration of the upper cranial nerves, chronic ophthalmoplegia and allied conditions, as being similar in character to chronic progressive bulbar paralysis, already described (page 436), and to the spinal form of progressive muscular atrophy. The transition between these lesions and the degree of acuteness of onset together make possible most varied symptomatic manifestations, of which a certain number stand out conspicuously, and have, therefore, been regarded as clinical entities. Among these chronic ophthalmoplegia is important.

CHRONIC OPHTHALMOPLEGIA.

In the great majority of cases chronic, usually progressive, ophthalmoplegia, forms a part of a more general process, as for example, tabes or dementia paralytica, and is frequently associated with progressive bulbo-pontine degenerations of motor nuclei, and at times with spinal cord lesions of similar character. As an early manifestation of one of these more extensive affections, as in those cases in which it dominates the clinical picture, chronic ophthalmoplegia is deserving of special consideration.

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[14] Taylor: Poliencephalitis and Allied Conditions, Boston Med. and Surg. Jour., cxlviii. 634, 1903.

The symptom-complex was first described by von Graefe. Its development is slow and its course long, in contrast to progressive bulbar paralysis, due to the fact that the process is located in a region of the brain not essential to life. Although varying widely in detail, the disease begins usually with diplegia or ptosis, followed by a very gradual extension of the palsy to the external ocular muscles of both eyes; the accommodative mechanism and the levator palpebrae superioris are often spared. Later the internal muscles may be involved, or in other cases they may be affected from the beginning. The progressive involvement of the muscles points always toward nuclear degeneration as a cause, easily explained by the complicated anatomical arrangement of the nucleus of the third nerve. As the process continues, the eyeball may ultimately become wholly immovable, and the levator palpebræ and orbicularis oculi also be involved. In rare cases the disease is checked when the ocular paralysis is complete (Strümpell). It is, however, usual for the degeneration to extend to other nuclei, with an ultimately fatal outcome.

Pathological Anatomy.—The usual lesion is a progressive degeneration of the cells of the nuclei of the third, fourth and sixth nerves, precisely analogous to that described under progressive bulbar paralysis.

PSEUDOBULBAR PARALYSIS.

Knowledge of this type of bulbar paralysis dates from 1872, when Joffroy first called attention to the fact that the symptoms of bulbar lesion may occur without disease of the oblongata, the essential alterations being in the cerebral hemispheres. An almost simultaneous publication by Jolly of a case with bulbar symptoms from cerebral multiple sclerosis, and in 1877 a report of cases by Lépine and Barlow established the essential identity of the symptom-complex. Lépine first used the term "pseudobulbar" as applied to these cases. Since that time papers have appeared by Kirchoff, Féré, Jolly, Oppenheim, and Siemerling, Russell and Taylor, Becker, Remak, Colman, Oppenheim, Bouchard, Karplus, Weisenburg, and others. From these various observations it is apparent that a bilateral destructive lesion of the cortico-bulbar tracts is sufficient to produce the general symptoms of bulbar

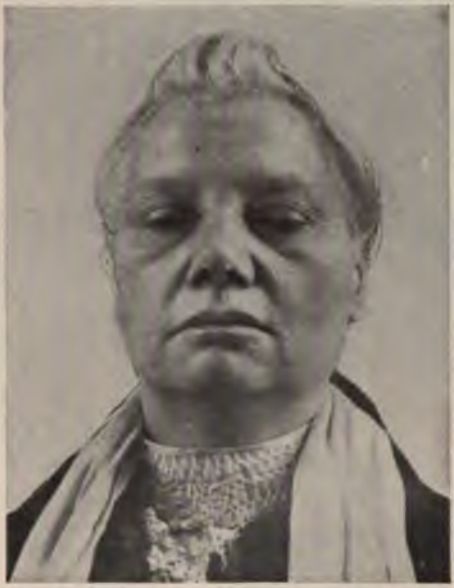


FIG. 7.—Poliencephalitis superior and inferior. The symptoms of gradual onset were dysphagia, paresis of soft palate, respiratory and cardiac disturbances, vertigo, somewhat exaggerated tendon reflexes (but no weakness of limbs), bilateral ptosis, intense external and internal ophthalmoplegia, diplopia, and pallor of optic disks. (Unreported case seen by Dr. Spiller with Dr. Thorington.)

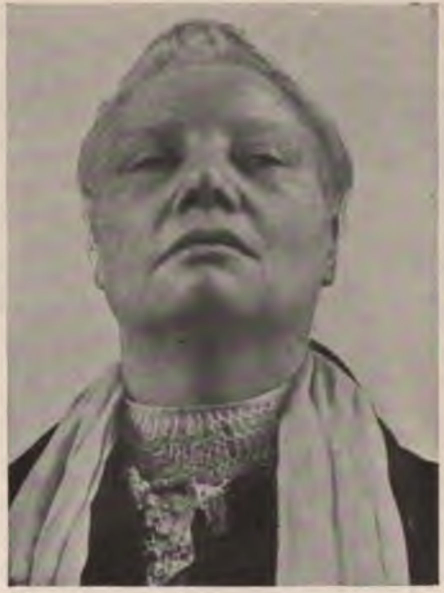
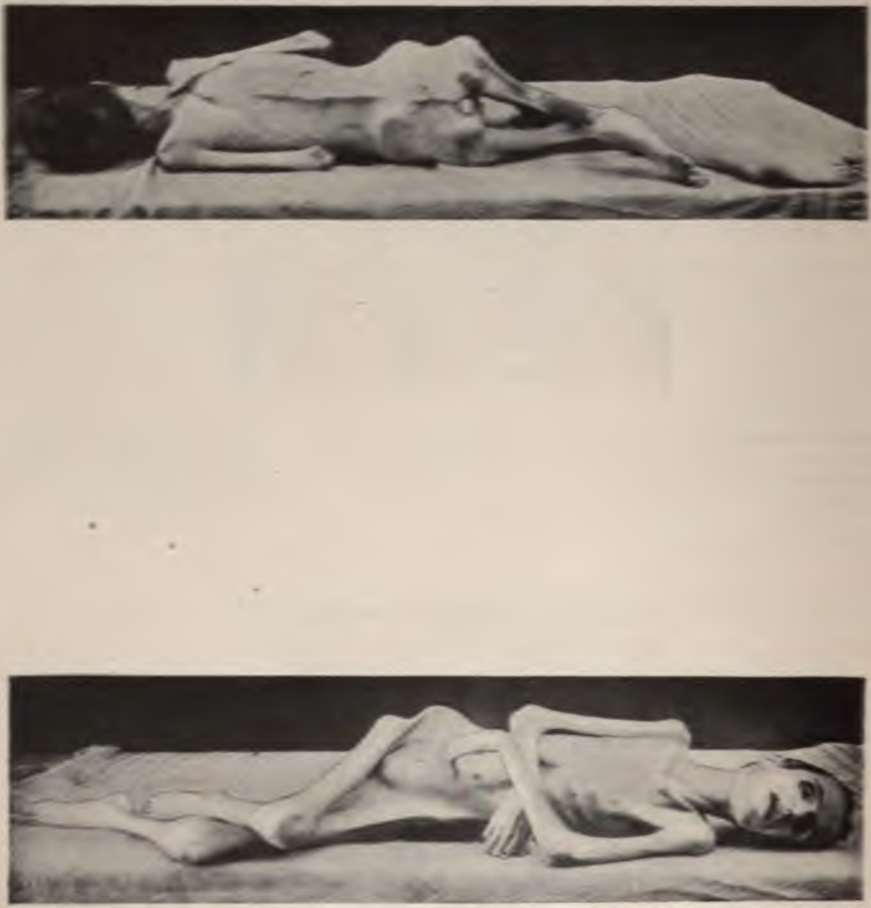


FIG. 8.—Same patient as Fig. 7. Photograph shows the bilateral ptosis and the inability to look upward.



Figs. 9, 10.—Intense muscular atrophy occurring in encephalo myelitis. (Case of Dr. Spiller.)

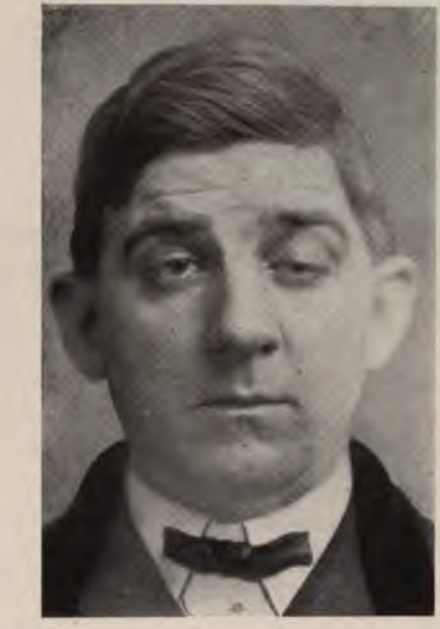


FIG. 11.—A case of myasthenia gravis, with paralysis confined to the ocular muscles. (Reported by Drs. Spiller and Buckman, Amer. Jour. of the Med. Sciences, April, 1905). The ptosis of the right eye is not so great as that of the left. The photograph was taken one or two minutes after glasses, one of which was opaque, were removed. At times the patient was able to open both eyes fully.

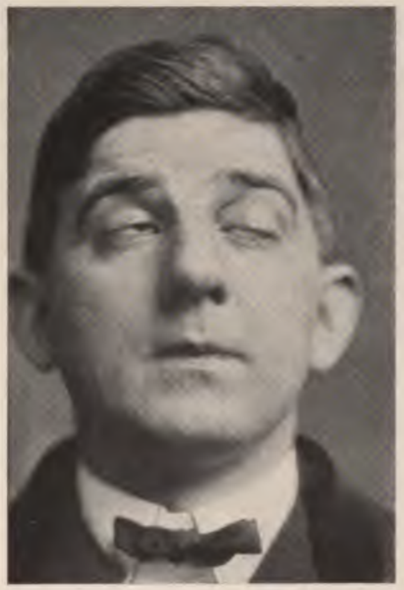


FIG. 12.—Same patient as shown in Fig. 11. The eyeballs are almost completely covered. The photograph was taken after the glasses had been removed three or four minutes.

palsy, presumably by a cutting off of the upper paths of voluntary control.

Pathological Anatomy.—The usual post-mortem findings are arteriosclerotic changes, with the resultant areas of softening and degeneration. The lesions are usually bilateral, though a few cases of bulbar palsy have been observed, in which the cerebral lesions were confined to one hemisphere. The usual locations of the softened areas are the cerebral white matter, internal and external capsules, central ganglia, particularly in the lenticular nuclei, and more rarely in the cortex. That such lesions are sufficient to produce the symptoms of bulbar paralysis has been shown by adequately studied cases. Oppenheim and Siemerling,[15] however, warn against the too hasty acceptance of cerebral lesions alone as explanatory of the symptoms. In the investigation of five cases, in which the pons and oblongata were studied serially, it was found that multiple small areas of softening were present in such a position as to require consideration in the production of symptoms. They very rightly maintain, therefore, that the cerebral origin of the symptoms should not be insisted upon until a careful microscopic examination of the pons-oblongata region is made. This apparently was not done in many of the earlier cases reported.

It seems advisable, therefore, to divide the cases on the pathological side into cerebral, and cerebrobulbar, recognizing the fact that a purely cerebral form is less usual than at first supposed. The cerebrobulbar forms evidently may not differ widely in their pathological anatomy from certain forms of acute bulbar paralysis, due to focal softening in the pons or oblongata, a general arteriosclerosis being the underlying condition in both cases. Oppenheim and Siemerling have also called attention to the importance of pressure exerted on the oblongata by a dilated, aneurismal or arteriosclerotic vertebral or basilar artery, in producing symptoms which might otherwise be attributed to more distant lesions. Recognizing these various possibilities, the following lesions are types of those which have been described as causative of the symptoms: bilateral softening in lenticular nucleus, parietal lobe, cortex of island of Reil, tempero-sphenoidal lobe (Féré); lesions in both capsules (Colman); cortical cystic tumors (Picot and Hobbs); cortical changes in an epileptic (Bouchard); diffuse lesions of hemispheres (Becker); microgyria (Oppenheim).

Symptomatology.—Considering the rather wide-spread lesions in the brain which are required to produce disturbance in the bulbar innervation, it follows that the symptoms are less sharply confined to this region than in processes before discussed. Various paralyses of the extremities, hemiplegia, paraplegia, together with aphasia, hemianopsia, and general mental defect, may form a part of the clinical picture, together with distinctly bulbar symptoms.

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[15] Oppenheim and Siemerling: Versammlung Deuts. Naturforscher, Berlin, 1886.

The development of the affection is characterized by repeated exacerbations, each leaving the patient with a further disability, and increased mental enfeeblement. Characteristic, and found only in this type, is progressive dementia, with various supervening conditions of confusion and excitement, forming part of the picture of a wide-spread arteriosclerosis. The paralyses of extremities are naturally dependent upon the position of lesions interrupting motor conduction. The significant symptoms for present consideration are those dependent upon lesion of the tracts to the bulbar nerves, whence comes the simulation of actual lesion of the oblongata. Loss of the power of speech, difficulty in deglutition, paresis of lower branches of the facial nerve (lip muscles) and of the palate, occur in a form superficially indistinguishable from true bulbar paralysis. The muscles involved are, however, not atrophied (disease of upper motor neurone) and the electrical reactions are not affected. Other symptoms of less constant occurrence are: Weakness of muscles of mastication; increased pulse-rate and arythmia; optic atrophy or neuritis; occasional impairment of bladder and rectal function; rarely anæsthesia in various localities. More frequent are respiratory disorders: attacks of dyspnœa and Cheyne-Stokes breathing. As in other forms of bulbar paralysis, uncontrolled expression of the emotions is frequent; due, no doubt, to a diminution of inhibition through destruction of paths of control, especially, it has been suggested, those from the cortex to the optic thalamus. Certain observations seem to show that a bilateral break in tracts from the cortex to the nuclei of ocular nerves may lead to disorder in movement of the eyes, of such a character that voluntary control is difficult, whereas they may still follow an object (Oppenheim and others).

The symptoms usually first manifest themselves after an apoplectic attack, often slight in itself, but succeeded at varying intervals by others, until finally the clinical picture is fully developed. Much more rarely the disease progresses slowly after an acute onset. The outcome is dependent upon the position, character and extent of the arteriosclerotic process.

It is evident in this, as in other inflammatory and degenerative processes, which have been considered in the foregoing pages, that an absolutely uncomplicated clinical type is rarely found. The distinction between cerebral and cerebrobulbar forms is often impossible to make clinically, and again the relationship between these forms and acute softening of the bulb is often confused. Many unclassified transitional forms of bulbar disease, dependent upon general arteriosclerosis, must be recognized, which it is not necessary to describe in detail, provided the underlying principle of the genesis of the symptoms be understood.

Prognosis and Treatment.—The outcome of pseudobulbar paralysis is ultimately fatal, through the increasingly disastrous results of the unchecked arteriosclerotic process. Remissions are frequent, but the general tendency is progressive, and little can be done by way of treatment, beyond a careful regulation of the patient's life in all particulars, and attention to the details of feeding and prophylaxis, as stated in a previous section.

MYASTHENIA GRAVIS.

This condition is also described under the following titles: Erb's disease; asthenic bulbar paralysis (Strümpell); asthenic paralysis; myasthenia gravis pseudo-paralytica (Jolly); bulbar paralysis without discoverable anatomical lesion; Erb-Goldflam or Hoppe-Goldflam symptom-complex.

Although an undoubted case was earlier described by Wilks, the credit of first calling definite attention to this affection belongs to Erb, who, in 1879,[16] on a basis of three cases described the group of symptoms, since known as myasthenia gravis. He drew special attention to the symptoms of ptosis, weakness of the muscles of mastication and of the neck muscles. No autopsy was obtainable in his one case which died; but a bulbar involvement was naturally suspected, from the character of the objective signs. Less importance was attached to coincident weakness of the tongue, muscles of deglutition and extremities. Muscular atrophy suggested a chronic poliencephalitis. In 1886 Jendrassik[17] published a probable case of the same affection, and in 1887 Oppenheim[18] called attention to a similar, if not identical, symptom-complex, which he then regarded as unique, apparently not considering Erb's earlier cases as coming into the same category. In Oppenheim's case a painstaking post-mortem examination showed no lesions, and the affection was, therefore, characterized as a chronic progressive neurosis manifesting itself essentially by the symptoms of a glossopharyngolabial paralysis without atrophy. Goldflam,[19] in 1891, more clearly defined the condition by laying stress upon the element of muscular fatigue as distinguished from actual paralysis, a matter later amplified by Jolly [20] in regard to electrical alterations, which he defined as the "myasthenic reaction." These changes consist essentially in a rapid tiring of the muscles under strong faradic stimulation, manifested by an increasing diminution in contractility.

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[16] Erb: Arch. f. Psych., ix. 336, 1879.

[17] Jendrassik: Arch. f. Psych., xvii. 301, 1886.

[18] Oppenheim: Virchow's Archiv, cviii. 522, 1887.

An important contribution to the general subject was made by Hoppe [21] in 1892, from Oppenheim's laboratory, in which he described a case of general motor weakness, but especially of the cranial nerves, of slow progression, in some regions going on to paralysis, with electrical alterations, but without atrophy; final fatal outcome and absolutely negative post-mortem findings. Hoppe considers this case not identical with Erb's, chiefly on the ground of complete absence of muscular atrophy. He further questions the utility of the term neurosis, as applied to the condition by Oppenheim, and regards it as probably of cortical origin.

Since this preliminary work, which established the disease on a fairly firm basis, a very large number of cases have been reported, with several critical reviews of the subject and ample bibliographical lists.[22] The general conception of the affection has not been materially altered by the work of the past few years, although a certain light has been thrown upon its possible pathological anatomy by Weigert,[23] and very recently by Hun and his collaborators, Drs. George Blumer and G. L. Streeter, and by Drs. C. W. Burr and B. F. Stahl.

Pathological Anatomy.—According to Hun's tabulation, autopsies in thirty-two cases have shown negative findings in the nervous system in seventeen cases. In the remaining cases lesions found in the nervous system were slight recent hemorrhages, and uncertain changes in motor nerves and nuclei of the oblongata, together with certain abnormalities of development, which cannot be brought into causal relationship with the clinical course of the disease. Findings outside the nervous system are probably of more significance; in fact, there is small evidence to show that a primary affection of the nervous system is in any way responsible for the symptoms supposed to be characteristic of the affection. Weigert's observation, made in 1901, is possibly of importance in the light of certain other similar findings, and especially of Hun's, to which allusion has been made. In a case of myasthenia gravis Weigert, in a routine post-mortem examination, found an extensive infiltration of the muscles with lymphoid cells, which he regarded as metastases of a malignant tumor of the thymus, of lympho-sarcomatous character. The appearances were so striking in this case that Weigert was inclined to think that had similar changes been present in all previous cases, they could not have been overlooked, and, therefore, that one is not justified in assuming that such a lesion is the constant cause of myasthenia. Hun has recently described a case identical with Weigert's, and on the basis of this fact, taken in conjunction with observations of similar character made by Link, Oppenheim and Goldflam, strongly advocates the theory that the secret of the pathological anatomy probably lies in these or closely analogous alterations. A very similar condition has recently been reported by Burr and Stahl before the Philadelphia Neurological Society, and Burr still further discusses the subject in the *Journal of Nervous and Mental Disease* for March, 1905. The final explanation of the muscular weakness and myasthenic reaction Hun seeks in a disturbance of the function of the muscle end-plates, brought about by interference with the lymph circulation in the muscles. To explain the cause of the infiltration of the muscles with lymphoid cells carries one wholly into the field of speculation and requires no discussion here.

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[19] Goldflam: Neurolog. Ctb., x. 162, 1891; also Deuts. Zeitsch. f. Nervenheilk., iv. 312, 1893.

[20] Jolly: Berl. klin. Woch., xxxii. 1, 1895.

[21] Hoppe: Berl. klin. Woch., xxix. 332, 1892.

[22] The more important of these are Campbell and Bramwell: Brain, xxiii. 277, 1900; Myers: Jour. of Pathol. and Bacteriol., viii. 306, 1903; Hun: Albany Medical Annals, xxv, 28, 1904 (124 references).

[23] Weigert: Neurolog. Ctb., xx. 597, 1901.

Symptomatology.—The disease is ordinarily insidious in onset, and characterized essentially by weakness and rapid tiring of muscles. In nearly one-half the reported cases, according to Hun,[24] ptosis is a very early symptom. In forty-five per cent. of the cases ocular palsy, either ptosis, or diplegia, occurred as the earliest symptom. In a smaller proportion, twenty-three per cent., involvement of the facial, hypoglossal and accessory nerves was the first symptom. A somewhat larger number of cases gave as the earliest sign of the disease weakness of the arms or legs or both. As the disease develops the usual characteristic picture is double ptosis, double facial paresis, paralysis of the bulbar nerves, associated with dyspnœa, and general bodily weakness. Among these symptoms the ocular palsies occupy probably the most conspicuous place, and of individual symptoms ptosis is the most frequent, occurring sooner or later in upward of eighty-five per cent. of all cases. Various external muscles of the eye are also frequently involved, but the internal muscles governing the pupil are very rarely included in the muscular weakness.

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[24] Hun's paper, loc. cit., contains a most valuable analysis of earlier cases in their various aspects.

Wilbrand and Saenger[25] in an analysis of forty-three cases find diplegia in fourteen; ophthalmoplegia exterior in nine; weakness of the sphincter iridis and ciliary muscles in one; unilateral ptosis in three; bilateral ptosis in twenty-nine; disturbance in the distribution of the facial nerve, a number involving the orbicularis oculi, in twenty-five. Gowers[26] has recently called special attention to the affection of the eye muscles in connection with a study of three personal cases. At first sight the ophthalmoplegia simulates the variety due to nuclear degeneration, but the differences are probably significant. In the myasthenic form, the muscles moving the eyeball downward are less involved than those moving it upward, the lateral muscles are constantly but irregularly involved, but differing in degree in associated muscles and also showing striking variations at different times. There is no correspondence in some of the cases between the degree of ptosis and the impairment of upward movements of the eyes. The orbicularis palpebrarum is often included in the weakness, and the frontalis may be. Gowers also draws attention to a characteristic facial expression, which he calls the "nasal smile," brought about by a failure of the normal movement of the corners of the mouth, the furrow caused by the attempt to smile being wholly confined to the upper lip, and ceasing towards the corners of the mouth. The cause of this peculiarity is a weakness of the levator labii superioris.

The involvement of muscles supplied by the facial nerve, in general, is a frequent sign, giving rise to so-called facial diplegia. The muscles of mastication, speech, deglutition and phonation may all be weak, and these disabilities, if conjoined with weakness of the neck, body, and extremity muscles, reduce the patient to a condition of practical helplessness. The face is expressionless, the eyes partially closed, the jaws are moved weakly, speech is impossible, swallowing is difficult, the head cannot be voluntarily supported, dyspnœa may come on, and the skeletal muscles be too weak to hold the body erect.

In spite of such a wide distribution of symptoms, the condition is in every instance due to a rapid tiring of the muscles, associated as the process develops with more or less persistent weakness. As a rule those muscles which are most constantly in use show the greatest weakness as a result of tiring, but this is by no means invariably the case. The order of involvement of the muscles is also not always as given above, beginning in the eyes, and extending gradually downward. The reverse may occur, and not infrequently the distribution of the weakness is wholly irregular.

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[25] Wilbrand and Saenger: Neurologie des Auges, 1899.

[26] Gowers: Clinical Lectures, Blakiston, 1904, Lecture on Myasthenia and Ophthalmoplegia, p. 221.

The association of weakness and abnormally rapid tiring of muscles on use must be regarded as an essential symptom of the disease. To this must be added the peculiar action of the affected muscles on electrical stimulation, first carefully studied by Jolly. This so-called "myasthenic reaction" consists in a rapid diminution in contractility when stimulated by a tetanizing faradic current. The galvanic current has a much less tiring effect according to most observers, although opinions on this point are not altogether uniform. Reaction of degeneration does not occur, and muscular atrophy forms no part of the clinical picture. The relation between voluntary and electrical (faradic) contraction of the muscles remains undetermined. Jolly found that exhaustion took place when the muscles were voluntarily contracted, in the same degree as when electrically stimulated; others have had a different experience. The myasthenic reaction, though not constant, occurs in some form in a large majority of the cases. In fatigue brought about by other causes, and in certain cerebral and spinal diseases, an electrical reaction similar to that occurring in myasthenia has been described, but the reaction, nevertheless, remains a characteristic and striking feature of the disease.

The sensibility is not disturbed in the affection, and the mental faculties remain unimpaired. The knee-jerks at times show a tendency to exhaustion; the sphincters are not involved. Naturally a great variety in the extent, grouping and degree of the muscular exhaustion leads to much diversity in the clinical picture. In general, the combination of cranial nerve weakness, and especially ptosis with accompanying weakness of the skeletal muscles; absence of muscular atrophy, with unaltered electrical reaction, apart from rapid tiring on faradic stimulation; tendency to variations from time to time; unimpaired sensibility and freedom from mental defect, form a symptom-complex which is found in no other condition.

Etiology and Course.—The disease ordinarily occurs in young adults, without reference to sex or hereditary influences. There is no uniformity in the various conditions which have been described antecedent to the actual appearance of the disease. Even if the finding of a lymphoid infiltration of the muscles with disordered lymphatic circulation be regarded as etiologically significant, the ultimate cause still remains obscure. To say that the affection is due to a toxæmia is rather to restate the question than offer a solution.

The onset of the disease is usually insidious, and its course slow, often with marked remissions followed by exacerbations. Successive groups of muscles are usually involved, often irregularly. Temporary improvement may take place for months or even years, with final relapse to the former or to a worse condition. Variability in the development of symptoms is so constant a feature of the disease as to be of great importance in the diagnosis. In ninety-eight of the tabulated cases the intensity of the symptoms varied from day to day and in sixteen only was the progression steady. A few cases have been described in which the onset was acute, and the disease fully developed in the course of a few weeks; these are, however, wholly exceptional. Death has terminated the affection in something less than fifty per cent. of reported cases; a small number, seven, are regarded as cured, and the others, fifty-seven out of one hundred and fourteen, are still living.

Treatment. The essential of treatment is the avoidance of muscular fatigue. In serious cases the patient should be kept in bed, and warned against exertion of any sort. Electricity should be applied with great care. Central galvanization is advisable. Oppenheim quotes a case in which a fatal outcome followed an attempt at artificial feeding. The food should be of such a character that the effort of swallowing may be reduced to a minimum; the possibility of sudden death from suffocation should be borne in mind. In milder cases less care is required, but the results hitherto attained are not of such a character as to warrant the hope that the course of the disease can be essentially modified. Appropriate tonic and general hygienic and symptomatic treatment will accomplish something toward the amelioration of the general condition.

PARALYZING VERTIGO (GERLIER'S DISEASE).

The disorder known as Paralyzing Vertigo, or Gerlier's Disease, was first described by a Swiss physician, Gerlier,[27] in 1887. Since that time he has contributed a series of articles, amplifying his first detailed description especially on the clinical side. David,[28] Haltenhoff,[29] Eperon,[30] Ladame,[31] and Sulzer[32] have made communications of less importance on the clinical aspect of the subject or in criticism of Gerlier's views.

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[27] Gerlier: Rev. Med. de la Suisse Romande, vii. 1, 260, 1887; idem, viii. 22, 86, 1888; idem, xi. 201, 260, 1891.

[28] David: Rev. Med. de la Suisse Romande, vii. 65, 1887.

[29] Haltenhoff: Prog. Med., xv. 515, 1887.

[30] Eperon: Rev. Med. de la Suisse Romande, ix. 38, 1889.

[31] Ladame: Critical Digest. Brain, xii. 534, 1890.

[32] Sulzer: Rev. Med. de la Suisse Romande, xiii. 695, 1893.

The affection has been observed in Europe only in Collex, a canton of Geneva, and in seven surrounding hamlets. Its victims are workers in the fields, and particularly cowherds. Vigorous men in the early period of life are attacked, during the summer months only, and those exposed to the worst hygienic conditions are usually the first and most seriously afflicted.

Symptomatology.—Three symptoms are characteristic; pain in the region of the neck, muscular weakness and ocular disorders. These may be variously combined, and occur with varying degrees of intensity in the same person at different times or in different persons. Vertigo, as ordinarily understood, is a less conspicuous symptom.

The neck pain is described as a sense of constriction, with a tendency to torticollis, the pain often radiating to the back. The muscular weakness is confined to those muscles normally under control of the will, and the extensors are more commonly affected than the flexors. The neck muscles are usually involved, but unsymmetrically on the two sides. The disturbance of the eyes is characterized by ptosis, usually unequal in the two eyes and of varying degree. This is not accompanied by strabismus, or palsy either of the external or internal ocular muscles, and there is at times a diminution in the visual fields. The fundus has apparently not been extensively examined, but so far as they have gone the ophthalmoscopic findings are negative, with the exception of an observation of Eperon, in which he found papillary hyperæmia and retinal hemorrhage, and a similar finding by Sulzer. A subjective sensation of clouding of vision, preceding the onset of ptosis, accompanied by diplegia, and vertigo, rarely circular in character, often occurs.

A typical attack may be described as follows: The patient, previously well, is suddenly attacked by pain in the neck and back; his sight is clouded to the point of temporary blindness; ptosis develops rapidly along with general weakness of neck and body muscles; standing erect becomes difficult; he reels and has all the appearance of drunkenness. After a period not exceeding ten minutes, complete recovery takes place. During an attack the tendon reflexes are preserved, at times increased; the skin sensibility is intact, false trismus occurs, swallowing is often impossible, and general motor weakness dominates the clinical picture. The head often falls forward on the chest, due to weakness of the neck muscles, which, with the ptosis cutting off vision, renders the patient for the time being wholly helpless. Nausea does not occur. Many of the attacks are very slight and merely momentarily incapacitate the patient, but the foregoing symptoms invariably are developed in some degree.

Etiology.—Nothing definite is known of the cause of the attacks. Gerlier was of the opinion, though it was not experimentally verified, that inasmuch as peasants working and sleeping in stables were usually affected, the cause was to be sought in an infection or "miasm," only active in the warm months of summer, derived from the soil of the stables. The malady has been shown to be seasonal, always checked by the onset of cold weather; the attacks, furthermore, occur usually with striking regularity in the afternoon, ceasing about an hour after sunset. They are, however, unaffected by the administration of quinine. Attacks are induced by excitement, brilliant light, by gazing fixedly at an object, or by the contemplation of a wide space. Ladame regards the affection as hysterical, and wholly due to suggestion. This Gerlier rather indignantly denies, giving as a conclusive argument the fact that he has observed the same attacks in cats, which presumably are not suggestible. Eperon is inclined to attribute the attacks to a meningeal hyperæmia, possibly allied to sunstroke; Sulzer considers the possibility of lesions of the occipital lobes, accompanied by descending neuritis of the optic nerves.

Miura in 1897 described a similar affection occurring in the northern provinces of Japan, to which he gave the name "Kubisagari." The clouding of vision he regarded as a nervous asthenopia, and the diplegia present in his cases as an insufficiency or paresis of the internal recti. The possible relationship of these conditions with asthenic bulbar paralysis, and also with the rare family periodic paralysis, should be borne in mind.

Treatment.—Iodide of potash, cold bathing, and generally improved hygiene have been recommended. The disease is never fatal, and is invariably terminated by the onset of cold weather.

CHAPTER XI.

ARTERIOSCLEROSIS; MULTIPLE SCLEROSIS; PSEUDOSCLEROSIS; DIFFUSE SCLEROSIS; FRIEDREICH'S DISEASE; PARALYSIS AGITANS.

By WILIAM HIRSCH, M.D.

ARTERIOSCLEROSIS.

According to our present views, arteriosclerosis forms merely a part of a general angiosclerosis,—*i.e.*, an affection of the entire vascular system, which may be due either to a general regressive metamorphosis, to the influence of toxic agents, or to congenital abnormalities in the structure of the vessels. Inasmuch as the integrity of every organ depends on its proper nutrition and blood supply, and this again is dependent on an unimpaired vascular system, it is evident that arteriosclerosis is apt to produce morbid changes in any and every part of the body.

During the earlier stages of arteriosclerosis the affections of the various organs consist merely of functional disturbances, such as irregularities in circulation are apt to produce. But as soon as the morbid changes in the blood-vessels become more marked, and consequently the circulatory disturbances more severe, the various organs are apt to become subject to organic diseases.

The functional disturbances which a general arteriosclerosis is apt to produce in the eye correspond to analogous symptoms in other organs. The muscles are easily fatigued and are lacking in endurance and power of resistance. Patients complain of difficulty in reading or of headaches after using the eyes, in spite of normal or by glasses corrected lenses. Besides these disturbances in accommodation and convergence, the diminution of muscular tonus may manifest itself by slight difficulties in moving the eyeballs, by incomplete and transitory ptosis, or by slight nystagmus, especially in horizontal movements of the eyes.

Arteries which are undergoing sclerotic changes are subject to such functional disturbances as spasm and temporary paralysis. The well-known symptom of a temporary scintillating scotoma is in all probability produced by a spasm of the blood-vessels supplying the cortical lobe. Such spasms may occur also in the blood-vessels of the retina, and the intensity and rapidity of the increasing pressure. The atrophy may be confined to one group of fibres, like the "crossed," the "non-crossed," or the "papillo-macular" fibres, and in some cases it might be possible to recognize approximately the seat of the pressure from the fibres involved in the atrophic process.

The prolongation of the bony part of the optic canal within the cranial cavity is formed by a ring of thick, fibrous connective tissue. This ring is covered by the dura mater, which near the optic foramen forms a tensely stretched free margin. The base of the intracranial portion of the canal is formed by the internal carotid artery. If, therefore, this artery becomes hardened and considerably enlarged in diameter, it may push both optic nerves upward and forward against the hard margin of the dura mater, crushing and destroying both nerves.

A sclerotic process in the carotid and in the vessels of the circle of Willis is, of course, apt to produce atrophy of one or both optic nerves by exerting pressure in the region of the chiasma. It is evident that eventually under suitable conditions the various forms of hemianopsia may be produced by a corresponding pressure.

So, then, we see that sclerosis of the cerebral arteries may affect the optic nerves in numerous ways. We may have partial or complete blindness in one or both eyes; we may have a narrowing of the visual field or central scotoma; we may have hemianopsia or amblyopia. Any of these conditions may occur during the course of a general arteriosclerosis, and most cases of so-called "spontaneous optic atrophy of old age" are evidently due to a sclerotic process in the cerebral arteries.

Arteriosclerosis has been regarded as a cause of quite a number of diseases of the eye. We will only mention these conditions here. Michel and his pupils have held that cataract can be produced by sclerosis of the carotid artery. According to some authors, glaucoma is produced by a general arteriosclerosis and the consequent disturbances in the circulation. Embolism of the central artery may be due to a sclerosis of vessels belonging to quite a different region of the body. A disease which only recently has been described more accurately, and which is supposed to stand in close relation to arteriosclerosis, is "retinitis circinata."

There can be no doubt that arteriosclerosis forms one of the most important etiological factors in the production of diseases in general, and the more our knowledge of this subject advances the more we will appreciate this important fact.

MULTIPLE SCLEROSIS; PSEUDOSCLEROSIS; DIFFUSE SCLEROSIS.

The optic nerve is affected in nearly 50 per cent. of all cases of multiple sclerosis, and, if we consider that the changes in the optic disk may constitute one of the earliest manifestations of the disease, or may even precede all other symptoms by several years, we will readily appreciate the great diagnostic value of the ophthalmoscopic findings in this disorder.

In order fully to understand the nature of the various affections of the optic nerve in multiple sclerosis, we must always bear in mind the fact that the optic nerve is not a nerve in the same sense as the other peripheral nerves, but forms a part of the brain, so that it would be much more appropriate to speak of the optic tract rather than of the optic nerve, even in its most peripheral parts.

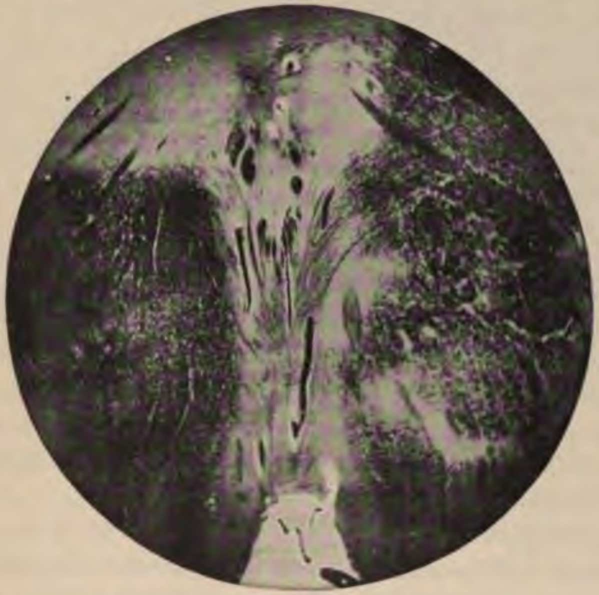

FIG. 1.—Multiple sclerosis, showing a sclerotic area in the oculomotor nucleus. (Case of Dr. Spiller.)

The optic nerve represents a system of neurones, and corresponds in every respect to other systems, such as the posterior columns or the pyramidal tracts, and its affections in the various diseases of the central nervous system are entirely analogous to the affections of the other systems. In locomotor ataxia, for instance, the system becomes affected as such, as a unit. The posterior columns degenerate as a system, and, if the optic nerve is affected at all, it degenerates also as a system, undergoing complete atrophy. Multiple sclerosis, however, is not a systemic disease. No system is affected as such. The multiple plaques are distributed indiscriminately through the entire brain and cord, regardless of any order or system. One plaque may reach from one system to the other, destroying a little here and a little there. The posterior columns may be affected at various levels, but they are not apt to be either more or less affected than any other system. According to this character of the disease, every system is more or less affected in multiple sclerosis, but no system is affected entirely throughout its course as is the case in the systemic diseases.

The optic atrophy in multiple sclerosis is, therefore, very different from that in locomotor ataxia. In the latter disease the affection is symmetrical, it progresses slowly but surely, and ends invariably in complete atrophy of both nerves. The atrophy is from the start equally distributed over the entire disk. In multiple sclerosis, on the other hand, the atrophy is hardly ever complete. There is either a partial atrophy distributed equally over the entire disk or a partial atrophy confined to its temporal half. The affection in a large percentage of cases remains one-sided. The morbid process may reach a certain stage and then remain stationary for a long time. The atrophic process never produces complete blindness in multiple sclerosis. On the contrary, an improvement has been reported in 50 per cent. of the cases. Occasionally even a *restitutio ad integrum* has been observed.

In some cases there was an optic neuritis either one-sided or bilateral. Sometimes the optic atrophy shows traces of a previous neuritis, so that it can be assumed that in a certain percentage the atrophy is a secondary condition to a primary neuritis.

The ophthalmoscopic finding often seems not to be in accordance with the real condition of the optic nerve. On the one hand there are cases in which vision seems to be considerably impaired in spite of an absolutely negative ophthalmoscopic picture, and on the other hand the disk may appear considerably atrophied and still the power of vision not be diminished in a degree which one would expect.

This peculiarity, as well as the other irregularities described above, can easily be explained by the anatomical nature of the disease. A plaque may destroy considerable substance of the nerve without affecting the parts in the immediate neighborhood of the retina, thus offering the clinical picture of a retrobulbar neuritis. On the other hand, an affection of the retinal part of the optic nerve does not indicate its entire condition, as it does in other cases of atrophy.

Of clinical significa [illegible] optic atrophy of multiple sclerosis is the irregular, atypical behavior of the visual field. There is no homogeneous narrowing, as in cases of tabes, but a most irregular constriction of the peripheral line. There may be a central scotoma or a combination of this with an irregular narrowing of the field. In a few cases an annular-shaped scotoma has been found, and in other rare instances small parts of the periphery were alone preserved while the function of all the rest of the nerve had been lost. Sometimes there is a central scotoma only for colors, or in some cases a general achromatopsia.

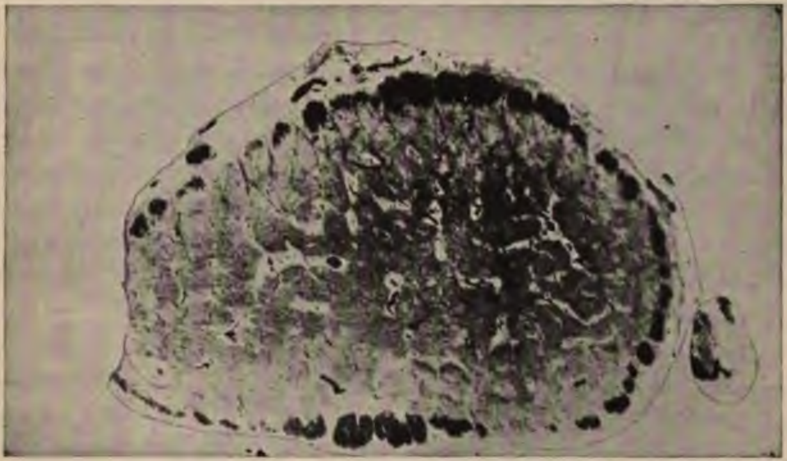

FIG. 2.—Multiple sclerosis of the optic nerve. (Case of Dr. Spiller.)

Paralysis of the muscles of the eye is a comparatively frequent symptom in multiple sclerosis, the various statistics showing an average of about 20 per cent. The nature of the paralysis is always that of a central lesion, characterized by a conjugate paralysis, paralysis of convergence, and the impairment of isolated muscles.

The paralysis itself is mostly an incomplete one, and its clinical course is characterized by manifold changes, such as improvements, exacerbations, intermissions, and relapses. The irregularity and the atypical course, therefore, correspond exactly to the pathological condition of the optic nerve. The latter differs, however, from the nerves of the eye-muscles by its frequent peripheral lesions, the other nerve-trunks being affected only very rarely in multiple sclerosis. Here again the optic nerve behaves like a part of the brain, differing also in this respect from all the true peripheral nerves.

Paralysis of the abducens seems to be the most frequent affection. Then follows the oculomotorius, which, however, is always affected only partially and incompletely; a complete paralysis of all branches of the oculomotorius, such as is seen in affections of the trunk of this nerve, hardly ever occurs in multiple sclerosis. Of the conjugate movements those to right and left are more frequently affected than the vertical movements of the eyeball.

There may be disturbances in the movements of convergence. In cases of this kind the recti externi appear impaired in this function, while in other functions, such as the conjugate lateral movements, they seem to be entirely intact. This shows very plainly the central nature of the lesion.

Ptosis has been observed in quite a number of cases, but, like the other affections, offers the character of incompleteness. It is more frequently unilateral than bilateral, and is often associated with double vision or other disturbances of sight.

Partial paralysis and disturbances in the function of the eye muscles may be produced by cortical, subcortical, nuclear, and fascicular lesions, according to the localization of the sclerotic plaques, and we must admit that in a great many instances we are still unable to localize the lesion accurately from its clinical manifestations.

A symptom to which a great diagnostic importance is generally attached in multiple sclerosis is nystagmus. Here we have to distinguish between the nystagmus proper, a condition in which the eyeballs are in constant motion, and the so-called "intentional nystagmus," or "nystagmus of motion," in which the oscillatory movements do not take place when the eyeballs are at rest, but only when they are turned towards an object, especially at the end of the horizontal movements. It is the latter form which we find most frequently in multiple sclerosis. Inasmuch, however, as slight oscillatory horizontal motions of the eyeballs after a sudden extreme lateral movement may occur also in hysterical or even normal individuals, we have to be very careful not to attach too much importance to this symptom, or at least confine its diagnostic value to the more marked cases. Sometimes the twitching of the eyeballs takes place only with the horizontal motions, while in other cases every motion, even vertical ones, may be accompanied by this phenomenon.

Quite a number of theories have been offered to explain this peculiar symptom of the disease, of which we will mention only the most important.

Some authors regard the nystagmus of multiple sclerosis analogous to the intentional tremor of the extremities, due to a paralysis of the centres of association or to an affection of the peripheral nerves. Others explain the nystagmus by a disturbance in the equilibrium, due to the insufficiency of certain groups of muscles. According to an other theory the nystagmus is produced by a diminution of the tonus of the muscles, the innervations from the centres being interrupted and impaired by the various sclerotic lesions.

Whatever the true cause of the nystagmus may be, so much is certain, that it is located in the central nervous system, in the brain or in the medulla oblongata. The various territories, which have been claimed to be responsible for this phenomenon, are still very numerous, and in all probability we have to deal here with a condition which can be brought about by manifold lesions in the brain.

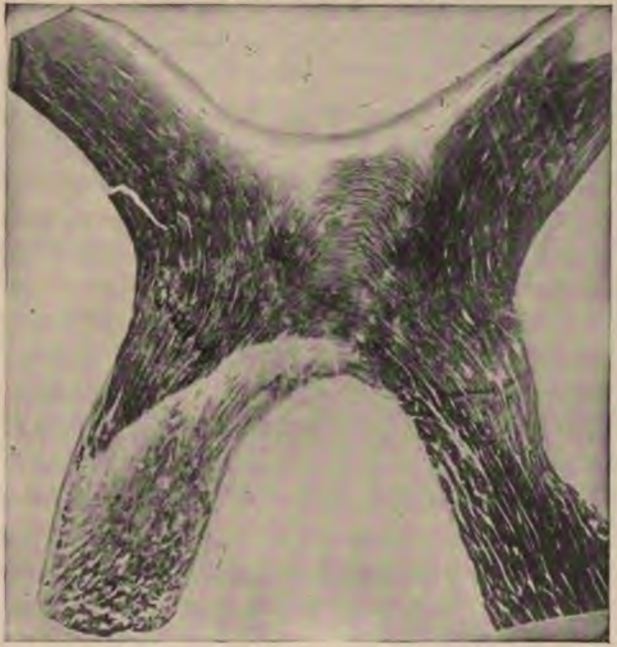

FIG. 3.—Multiple sclerosis of the optic chiasm and one optic nerve. (Case of Dr. Spiller.)

The function of the pupils remains normal, as a rule, throughout the entire course of the disease. If, however, we bear in mind the anatomical picture of multiple sclerosis, if we consider that sclerotic plaques are apt to occur in any part of the central nervous system, we will readily understand that there is really no symptom in neuropathology which may not occasionally occur in this disease. Abnormal conditions of pupils, therefore, have been reported, but they really do not belong to the characteristics of the disease, and if they do occur, they mislead rather than assist in diagnosis. The pupillary symptoms, which were observed in very rare cases, were: myosis, difference of pupils, diminution and exaggeration of light-reflex, loss of light-reflex, loss of reflex on accommodation and paradoxic reaction.

There is hardly any other disease of the central nervous system in which the clinical phenomena of the eye are so characteristic of the pathological process, and in which, therefore, the symptoms are of the same significance as in multiple sclerosis.

There is a disease, the clinical manifestations of which resemble multiple sclerosis, but wherein the anatomical changes do not correspond to the clinical symptoms. This affection was first described by Westphal under the name of pseudo-sclerosis, and recently Struempell has thrown more light on this peculiar subject. It is of special importance to us, inasmuch as the condition of the eye forms one of the main factors in the differential diagnosis between this affection and genuine multiple sclerosis. While the other symptoms offer a great resemblance to the clinical features of multiple sclerosis, the eye symptoms always remain absent in pseudo-sclerosis. There is no optic atrophy, no muscular palsy, no nystagmus.

Another affection, which offers many clinical resemblances to multiple sclerosis, pseudosclerosis and general paresis, is called diffuse cerebral sclerosis. This is a disease which may occur at all ages, in children as well as in adults. The main symptoms consist of spasticity of lower and upper extremities, increased reflexes, mental apathy and dementia and various palsies which may increase to complete paralysis. This disease also is characterized by the absence of any ocular symptoms, optic neuritis having been observed only in a single case (Heubner).

FRIEDREICH'S DISEASE.

In spite of the general interest which was created by Friedreich's first publications and which has prevailed ever since, in spite of the numerous excellent clinical observations and anatomical investigations of this disease, we are still far from having reached a generally accepted view regarding the nature and pathology of this affection. Even with reference to the clinical features of the disease, there still exist great differences of opinion, especially as to the grouping into various types.

As far as ocular symptoms were concerned, it was emphasized by Friedreich himself, that the disease is characterized by a complete absence of any abnormalities or disturbances in the eyes, with the exception of one symptom, namely, a certain form of nystagmus.

This nystagmus, which is sometimes called pseudo-nystagmus, or atactic nystagmus, does not consist of the rhythmical oscillations of the eyeball like the ordinary nystagmus, regardless whether the eyes are at rest or whether movements of some kind are performed; it consists of irregular twitchings, which occur only when the eyes are fixed on a moving object, especially in the horizontal direction. When the eyes are at rest, or when they are fixed at a point in the sagittal plane, they are as a rule perfectly quiet. This form of nystagmus, which resembles that of multiple sclerosis, only being more intense than the latter, forms one of the most constant phenomena of Friedreich's disease. Only very rarely is this symptom absent; but then it can sometimes be produced artificially by turning the patient several times around his axis. In all probability this phenomenon has to be considered as a disturbance in co-ordination, as an atactic condition of the muscles of the eye.

As said before, this was claimed by Friedreich to be the only ocular symptom of the disease, and up to the present day a great many authors regard all other abnormalities of the eyes as not belonging to this affection, and therefore as a contraindication to the diagnosis. Inasmuch, however, as soon afterwards a considerable number of cases were reported, which did offer ocular symptoms and differed also in other respects from the cases described by Friedreich, Pierre Marie described another clinical picture under the name of *hérédoataxie cerebelleuse* (hereditary cerebellar ataxia). This disease, which had a great many traits in common with Friedreich's disease, such as the familiar character, the ataxia and progressive nature, differed from it by the condition of the patellar reflexes, which were increased here and absent in Friedreich's disease, and also in the symptoms of the eye, muscular palsies, loss of pupillar reflex, disturbances of vision, various affections of the optic nerve being comparatively frequent in this disease.

A theory had been offered according to which Friedreich's disease consisted of a maldevelopment of certain parts of the spinal cord, while the affection described by Marie was due to an abnormal cerebellum.

The attempt to keep these two types of the disease apart has not been successful. The more observations were put on record, and the more attention was given to this interesting disease, the more it was realized that such a division is more or less arbitrary. It soon became necessary to regard some cases as a "mixed type," as they offered features of each group in equal proportion. Some cases were diagnosed as Friedreich's disease by one observer and as cerebellar ataxia by another. In a few instances both diagnoses had to be made among brothers, one resembling the one and the other the other type.

Even anatomically it was not possible to keep the two types apart. In some cases both the cerebellum and the spinal cord were affected. In others the anatomical findings seemed not to correspond to the clinical features. According to one theory the disease always consists of a spinocerebellar affection, the tracts communicating between the cord and the cerebellum in both directions being affected.

The division of the disease into these various types will in all probability not withstand future investigations. All that we can say to-day is, that clinically as well as anatomically the features of the disease may vary, and that we must not draw the line too closely in making the diagnosis, but rather allow a certain latitude, even if the clinical picture differs from the original description of Friedreich.

As far as the eye-symptoms are concerned we may say that the special form of nystagmus, which was described above, is by far the most frequent and constant symptom of the disease, but that, in some cases it may be absent. Ocular palsies, optic neuritis, and even the Argyll-Robertson pupil may occur in the disease, although they are comparatively rare.

PARALYSIS AGITANS.

Ocular symptoms do not form an essential feature of paralysis agitans. There are a few cases on record in which paralysis agitans was combined with other diseases of the central nervous system, as, for instance, locomotor ataxia. The loss of pupillar reflexes or affections of the optic nerve in cases of this kind would, of course, not form a feature of paralysis agitans, but of the complicating disease.

Inasmuch, however, as in paralysis agitans the muscular apparatus of the entire body is apt to be affected in the characteristic manner, it is evident that the muscles of the eye may once in a while show the same morbid condition, although as a rule they escape even this affection.

The characteristic condition of the muscles consists of the typical rhythmical tremor, and of a very marked tension, which interferes with normal motion, so that in the later stages the individual may become nearly unable to perform any movements at all, although there is no true paralysis in the ordinary sense. The main difficulty in performing motions adequate to their purpose seems to arise from the inability of the patient to contract a group of muscles suddenly after they are at rest, or to relax them quickly when they are contracted. Both these functions are indispensable to normal motion. This condition would also explain the inability of the patient to arrest the body suddenly when in locomotion, a symptom known by the names of propulsion, retropulsion and lateropulsion. If a person, who is walking, wants to arrest this locomotion suddenly, he must quickly contract the antagonistic muscles, must throw the trunk in the opposite direction, a function which an individual afflicted with paralysis agitans is unable to perform.

If now we apply this condition of the muscular apparatus to the muscles of the eye, we would expect quite a number of interesting phenomena. To the tremor would apparently correspond a nystagmus. But this symptom has never been observed in paralysis agitans. This fact furnishes another proof for the assumption that nystagmus is produced by some central lesion and not by a disturbance in the general muscular tonus, because if the latter view were correct we would be bound to observe this symptom at least occasionally in paralysis agitans.

The tension and rigidity do occur in the ocular muscles, although far less frequently than one would expect. The sphincter is never affected; the pupils have always been found normal in this disease. Likewise the optic nerve always remains unimpaired. The rigidity is very apt to show itself in the orbicularis, thus interfering with the mimical motions, the expression of the features, and therefore contributing to the mask-like appearance of the face. The closing and opening of the eye is sometimes very slow and sluggish, and in a few cases there was a partial ptosis. In order to assist in the opening of the eyes, the musculi frontales are contracted, and on account of the difficulty in relaxing the muscles, patients often have marked wrinkles in the forehead.

The movements of the eyeballs are sometimes somewhat retarded. A few cases have been observed where there was great difficulty in convergence; also a spasm of accommodation has been described. In one case Graefe's symptom occurred on one side in the hemilateral form of the disease.

There is only a single case (Minkowski) on record in which there was a complete ophthalmoplegia externa. Both eyes were entirely closed and could not be opened by the patient. When one raised the lids with the finger, the eyeballs stood in a parallel axis, directed straight forward, without ability on the part of the patient to move them in any direction.

CHAPTER XII.

PARASYPHILITIC AFFECTIONS, INSANITIES, AND TOXIC ENCEPHALOPATHIES.

By F. X. DERCUM, M.D.

PARESIS.

The ocular phenomena of paresis consist in disturbances which may involve all parts of the visual apparatus,—namely, the muscles, internal and external, the optic nerve, and the fundus. These disturbances must be studied in detail, but, in order that we should do so intelligently, it will be necessary, first, to enter upon certain general considerations. Paresis is an affection which begins, as a rule, insidiously and extremely gradually. Its earliest symptoms are usually those of a purely functional disturbance, and occasionally this disturbance not only precedes the appearance of physical signs, but antedates the latter by a considerable period of time. During this period the symptoms may, roughly speaking, resemble those of neurasthenia, and the differential diagnosis between the two affections is attended with considerable difficulty and may indeed be open to serious doubt. Paresis, we may here remark, is readily divided, as regards its course, into three stages,—first, the initial or prodromal period; second, the period of the fully developed disease, and, third, the period of profound dementia—the terminal period. During the second or third stages no difficulty with regard to diagnosis can possibly arise as each of these stages is attended by physical signs, both positive and numerous. It is in the first stage only, as just stated, that difficulty may arise. If, therefore, the existence of an early physical sign can be determined, a vast assistance is given to the problem. Indeed, the appearance of such a symptom assumes a decisive importance.

Paresis, we should remember, is a degenerative disease attended by a constantly increasing loss of function. One of the cardinal points to be borne in mind in its early diagnosis is that the changes in function are quantitative and not merely qualitative. In its very beginnings are these fundamental features to be noted. There is a general though slight mental enfeeblement, an inability to apprehend readily, a faint but unmistakable loss of memory, slight lapses in conduct, and a neglect of the proprieties. There is especially an evident inability of the patient to properly appreciate his surroundings; his close contact with his environment is lost, and this is true not only as regards his relation to matters of business and to his family, but also as regards his own illness. This blunting or obtusion of the mental faculties is the early and often faint expression of the more profound dementia to supervene in the future. In neurasthenia, on the other hand, the character of the mental operations never changes; but one cardinal symptom is noted,—namely, the diminution in the capacity for sustained mental effort. There are never, however, any losses of function. There is no loss of memory, no blunting of judgment, no obtusion of the moral and æsthetic faculties—none of those countless changes in conduct and habits to indicate a progressive psychic disintegration.

Notwithstanding the fact that such fundamental differences obtain in the symptomatology of neurasthenia and of early paresis, it is at times a matter of extreme difficulty in practice to arrive at a decision, so faint and shadowy may the beginning symptoms be. It is obviously of vast importance to discover actual physical signs at the earliest moment. Unfortunately, it happens that at this stage the pupils and eye-grounds often fail to yield us any information. One sign, however, that may be present and one which I personally regard as of great significance, is the existence of a slight degree of amblyopia. With or without a diminution of the color sense, it is an invaluable symptom. It may antedate distinct and demonstrable anomalies of the pupils or changes in the eye-grounds. Visual hypæsthesia is apparently in keeping with the general psychic obtusion that is setting in, as well as with the lessening of general cutaneous sensibility noted sooner or later. It is a sign whose importance is not sufficiently recognized, nor is it sought for in individual cases as it should be. The difficulty with regard to light perception is not always slight in the beginning, but may amount to an actual loss of vision, though this early loss is frequently transient in duration. Further, it is admitted by numerous writers—Mendel, Mickle, Kraepelin, Ziehen, Ballet, and Blocq—that optic atrophy may be the first noticeable symptom of paresis.

The most common of the ocular phenomena of paresis are those which relate to the pupil. One of the earliest phenomena usually observed is that of inequality of pupils. Difference in the size of the pupils, we should remember, may occur physiologically, but in such case this difference is usually slight. If, however, a difference in the size of the pupils be noted, and this difference be associated with a sluggishness to light reaction or if the pupils, being equal, react slightly differently to light, the finding is suggestive with regard to paresis in its incipiency. In the early stages of paresis, indeed, a difference in the promptness of the response to light reaction between the right and left pupils is almost the rule. Likewise an impairment of the consensual light reaction may be the first symptom of a pupillary disorder in paresis, and it is of the same value as a difference of reaction of the two pupils.

The above facts are established by numerous observations and admit of no question. How valuable slight changes of the pupils are in enabling us to make a diagnosis is revealed by a case reported by Lucas in which a diagnosis of melancholia had been made, but in which Joffroy afterwards noted inequality of the pupils and suspected a paresis, which later matured.

Narrowing or small size of the pupils, even if slight, taken together with other symptoms, is very significant. Distinct myosis is, of course, a symptom of unquestioned value. Exceptionally, it occurs in the very early stages of paresis. Changes in the fundus of the eye may also be noted early, though such changes are exceptional. However, they have been noted early by Walker and more especially by Knapp. Knapp, indeed, has reported three cases of paresis in which optic atrophy preceded the mental symptoms. Abolition of the light reflex with preservation of accommodation—the Argyll-Robertson pupil—may also infrequently be observed in the early stage of paresis, though this is quite unusual. Commonly, sluggishness of the light reaction alone is noted in the early stage.

The mental changes of paresis may long antedate the appearance of physical signs. However, this is not by any means the rule. Physical signs may indeed make their appearance either simultaneously with the mental changes or may sometimes even precede the latter. Notably is this the case with the visual phenomena. In other words, it is unusual, in practice, to meet with early cases of paresis in which some ocular changes, even though slight, cannot be found.

The relative frequency of ocular phenomena in the early part of the disease may be gathered from the statistics of Joffroy. Joffroy examined two hundred and twenty-seven cases of paresis, of which the majority were in the first period of the disease. Two hundred and twelve of these cases presented ocular symptoms. Joffroy believes that among the fifteen who did not have ocular symptoms a later examination, at a more advanced stage, would certainly have revealed them.

If a systematic study be made of the eyes in paresis in the stage of the well established disease, the following phenomena are noted. First, there is very frequently observed an inequality of the pupils.

Some idea of the relative frequency of this symptom can be gained by the following statistics which have been taken in part from the groupings of Mignot and of Bumke. Siemerling noted inequality in 26 per cent. of the cases examined by him, Lasegue 33 per cent., Francotte 51 per cent., Moreau 58 per cent., Mendel 60 per cent., Mobêche 61 per cent., Kaes 62.7 per cent., Marie 63 per cent., Oebecke 64 per cent., Renaud 65 per cent., Seifert 66 per cent., Boy 66 per cent., Regis 66 per cent., Doutrebente 77 per cent., and Vincent 80 per cent. Mignot found 64 per cent.; Cololian, who studied fifty-seven cases, found unequal pupils in almost all of them. Dawson found 92.3 per cent. in the forty cases studied by him. It was the most frequent of all the ocular phenomena in his cases. The right pupil was the larger in twenty-five, the left in eleven. Kornfeld and Bikeles found unequal pupils in 70 per cent., Raehlmann in 60 per cent., Gudden in 57.5 per cent., Wallenberg in 53 per cent., A. Westphal in 45 per cent., Hillenberg in 45 per cent., and Siemerling in 27.5 per cent. Joffroy found inequality of the pupils in one hundred and forty-four of his two hundred and twenty-seven cases—that is, 63.4 per cent. If to these there are added his cases of extreme double mydriasis and extreme double myosis, he obtains 87 per cent. of cases of modifications of the dimensions of the iris.

Variations in the frequency with which unequal pupils are observed is, in part, to be attributed to the stages at which the cases have been examined and to a less degree to the element of personal equation: some observers noting minute differences and others only inequalities that are striking. Notwithstanding the divergence in the percentage obtained by different observers, it cannot but be admitted that inequality of pupils is an exceedingly common symptom of paresis. However, of itself inequality of the pupils has no more than a confirmatory value in the diagnosis. As is well known, it may occur in gross organic disease, brain syphilis, and the insanities; indeed, it may in moderate degree be occasionally noted in perfectly normal individuals. It is important to add that the degree of inequality in paresis may vary decidedly in a given case, and sometimes during a comparatively short period of observation. Rapid fluctuations are by no means infrequent.

The pupil in paresis is not infrequently irregular in shape,—that is, it may be slightly oval or ovoid or its circumference may be made irregular by various modifications of shape. These modifications do not, however, lead to gross deformities; notwithstanding, they can be very readily observed. They are undoubtedly present in a percentage of cases. Kaes found them in 3.1 per cent., Mobêche in 61 per cent., and Mignot in 18 per cent. Dawson noted irregularity in outline in one or both pupils in thirty-seven out of thirty-nine cases (94.8 per cent.). Joffroy noted in one hundred and twenty-five cases, in which the symptom was studied, deformity of both pupils in ninety-three,—that is, 75.2 per cent. In eight cases one pupil only was deformed. Mignot observed deformities in fifteen cases out of twenty-two. In seven of his cases Mignot observed the gradual diminution and final loss of the pupillary reflexes. Joffroy observed irregular pupils in two cases in which the symptom was not accompanied by impairment of the light reflex. However, in these two cases he later observed, first, paresis and then paralysis of the light reflex. Beginning deformity or irregularity of the pupil is to be regarded as a precursor of the Argyll-Robertson sign. In one case inequality made its appearance while the patient was under observation at the hospital. In another, pupillary disturbances made their appearance fifteen days after admission.

Myosis is also a symptom very frequently observed in the well-established period of the disease. The opposite condition, mydriasis, likewise occurs, and would appear from the following figures to be decidedly less frequent than myosis.

Kaes found myosis in 7.2 per cent., Mobêche in 12 per cent., Doutrebente in 12 per cent., Moreau in 18 per cent., Vincent in 38 per cent., and Marie in 39 per cent. Mignot found, out of twenty-two cases, myosis in six. Dawson found myosis on both sides in six cases out of twenty-three; in three cases he noted myosis in only one eye.

As regards mydriasis, Kaes found it in 3.6 per cent., Doutrebente in 4 per cent., Vincent in 14 per cent., Mobêche in 15 per cent., Moreau in 26 per cent., and Marie in 44 per cent. Out of twenty-two cases Mignot found mydriasis in only one. Out of twenty-three cases Dawson found mydriasis in eight. In one case he observed mydriasis of the right eye and myosis of the left. Mydriasis is a paralytic phenomenon which Fournier regards rather as a symptom of syphilis than of paresis. That it should, at times at least, be regarded as an expression of ophthalmoplegia interna and indicative of nervous syphilis rather than of paresis seems plausible, but involvement of the intrinsic muscles of the eyes is so frequent in cases of undoubted paresis that this explanation can apply only exceptionally—perhaps in cases in which true syphilitic processes are going on at the same time as the degenerative changes of paresis.

Next most important are changes in the light reflex. This is impaired or lost upon one side or both in a very large number of cases. In statistical studies of the loss of the reflex, different observers have likewise arrived at different percentages of results, as the following figures (grouped by Mignot) will show: Oebecke 26 per cent., Thomsen 47 per cent., Mendel 47 per cent., Siemerling 54 per cent., Magnan and Serieux 60 per cent., Moeli 61 per cent., Siemerling (second statistic study) 64 per cent., Francotte 88 per cent., Siemerling (third statistic study) 68 per cent., and Vincent 90 per cent. To these should be added Dillenberg 47.5 per cent., Wollenberg 50 per cent., A. Westphal 50 per cent., Gudden 41.2 per cent., Kornfeld and Bikeles 62 per cent., and Hirschl 50 per cent. Joffroy noted loss or impairment of the light reflex in one hundred and seventy-one cases out of the two hundred and twenty-seven cases studied by him,—that is, in 75.3 per cent. It was abolished on both sides in one hundred and three cases, abolished upon one side and diminished upon the other in fourteen cases, diminished upon both sides in thirty-five cases, abolished upon one side and normal upon the other in nine cases, and diminished upon one side and normal upon the other in ten cases.

M. de Montyel has made an exhaustive analysis of the light reflex. He found it normal in 30.8 per cent., abnormal in 69.2 per cent. Exaggeration was present in 5.3 per cent., diminution in 43.1 per cent., loss in 47.6 per cent. Analyzing his findings with regard to the period of the disease, he found the reflex exaggerated in 17.4 per cent. during the first period, and 2.3 per cent. in the second period, and never during the third period. He found it diminished in 44.6 per cent. in the first period, 47.2 per cent. in the second period, and 34 per cent. in the third period. He found it lost in 32.5 per cent. in the first period, 45 per cent. in the second period, and 66 per cent. in the third period. He found it normal upon one side and lost on the other in 5.5 per cent. in the first period, 5.5 per cent in the second, and never in the third period. Analyzing his results with regard to the form of paresis, he found it abnormal in 66.1 per cent. in the expansive form, in 100 per cent. in the depressive form, in 45.4 per cent. in the mixed form, in 80.6 per cent. in the simple demented forms, and in 25 per cent. of cases with remission.

Dividing his cases into those which were agitated and those which were quiet, he found it abnormal in 69.2 per cent. in the agitated forms and 75.8 per cent. in the quiet forms. Analyzing his results again as to ages, he found the light reflex abnormal in 93.8 per cent. in the ages from twenty to thirty, in 63.9 per cent. in the ages from thirty to forty, in 67.2 per cent. in the ages from forty to fifty, and 70 per cent. in the ages of fifty to sixty. De Montyel's conclusions from these studies were as follows: First, the light reflex was more frequently abnormal than normal. Second, exaggeration of the reflex was exceptional. Third, there was only a slight difference in the frequency of the occurrence of the diminished and the lost reflex. Fourth, the changes in the reflex were identical on the two sides; only exceptionally was the reflex lost upon one side and normal upon the other. Fifth, in the first period the normal reflex was more frequent than the abnormal, while the reflex is more frequently abnormal in the second and third periods. Sixth, exaggeration and unequal changes on the two sides were always present in the initial stages of the disease; the frequency of loss was proportionate to the progress of the disease. Diminution was more frequent than loss during the first period; the proportion was about equal in the second and reversed in the third period. Seventh, the reflex was abnormal in one-fourth of the remissions. Eighth, exaggeration was seen only in the expansive and mixed forms. Loss of the reflex prevailed over diminution in the demented form; only in the demented form were both eyes unequally affected. Ninth, an abnormal reflex was always noted in the traumatic cases. Tenth, at all ages, the abnormal reflex was more frequent than the normal; however, abnormality of the light reflex was especially noted in the extreme ages, especially in the younger cases. Eleventh, in the first and second periods the reflex was more affected when the motor symptoms were more pronounced. (See Chapter VI, p. 308).

The consensual light reflex should also be studied. Its importance is not fully appreciated. Dawson found absence of the consensual light reflex in one or both eyes in twenty-seven out of forty cases (67.5 per cent.). Further, it was involved without impairment of the direct light reflex in eleven cases, though in only two of these cases was it actually absent. Impairment or loss of the consensual light reflex must be regarded as of great significance whenever met with. Dawson concludes that it is more often affected than the direct light reflex.

Next, it is of importance to study the reaction to accommodation. This is preserved, as in tabes, long after the light reflex is lost. However, it is sooner or later involved. The more advanced the case, other things equal, the more likely is loss or impairment of accommodation to be present. Mignot, in twenty-two cases, found accommodation to be normal in eleven, lost in five, and diminished in six. De Montyel made an elaborate study of the reaction in thirty cases. These cases were followed from the outset to the termination of the disease, the reflexes being examined, in all, some six hundred and eighty times. It was found normal in two hundred and eighty-one examinations, abnormal in three hundred and ninety-nine. In fifteen examinations, diminution was present in one hundred and sixty-eight and loss in two hundred and ten. It was normal on one side and abolished on the other in six examinations.

In almost all the cases changes were present in both eyes simultaneously. Only in 1.5 per cent. was the reflex normal upon one side and lost upon the other. The majority of cases presented a diminution of the reflex. When exaggeration was present it was always moderate. Only six times did the reflex show changes in one eye. Six times the loss of of the reflex of one eye coincided with abnormality of the other. Analyzing his results as to the various periods of the disease, de Montyel found that in the first period the accommodation reflex was normal in 63.2 per cent., abnormal in 36.7 per cent. During the second period these figures were reversed, for now he found them normal in only 33.7 per cent. and abnormal in 66.3 per cent. In the third period a still greater reversal of percentage took place. It was normal in 16.7 per cent. and abnormal in 83.2 per cent. He found it exaggerated in the first period 15.3 per cent. He at no time found it exaggerated in the second or third periods. He found it diminished in 64.2 per cent. in the first period, in 41.9 per cent. in the second period, and 26.1 per cent. in the third period. He found it lost in 20.4 per cent. in the first period, 54.4 per cent. in the second period, 73.9 per cent. in the third period. In 3.5 per cent. he found it normal upon one side and abolished on the other in the second period of the disease. Analyzing those cases in which the reflex was found to be merely diminished and not lost, he found that it was somewhat diminished in 76.2 per cent. and very much diminished in 23.8 per cent. in the first period. During the second period he found that it was diminished in 88.6 per cent. and much diminished in 11.4 per cent. In the third period he found it diminished in 82.9 per cent. and much diminished in 17.1 per cent. Analyzing his results according to the depressive, the expansive, or the mixed forms, or forms with the remissions, he found the reflex normal in 53 per cent. and abnormal in 46.9 per cent. in the expansive form, normal in 51.7 per cent. and abnormal in 48.3 per cent. in the depressive form, normal in 53 per cent. and abnormal in 46 per cent. in the mixed form, and in the demented form normal in 25.3 per cent. and abnormal in 74.7 per cent. While in the cases with remissions he found this reflex normal in 62.5 per cent and abnormal in 37.5 per cent. He also found that it was exaggerated in 15.2 per cent. in the expansive form and never exaggerated in the depressive, mixed, simple demented forms, and forms with remissions. It was diminished in 42.4 per cent. in the expansive form, in 100 per cent. in the depressive form, in 41 per cent. in the mixed form, and 33.3 per cent. in the simple demented form, and in 100 per cent. in the forms with remissions. It was lost in 42.4 per cent. in the expansive form, never entirely lost in the depressive form, lost in 58.8 per cent. in the mixed form, in 64 per cent. in the demented form, and never entirely lost in the form with remissions. It was normal upon one side and abolished on the other in no case of the expansive, the depressive, or the mixed form. This, however, occurred in 2.5 per cent. in the simple demented form, and in none of the cases with remissions.

Analyzing his findings as to age, de Montyel found that the reflex was abnormal in 88.9 per cent. in the ages from twenty to thirty, in 57 per cent. in the ages from thirty to forty, in 21 per cent. in the ages from forty to fifty, in 82.9 per cent. in the ages from fifty to sixty. His conclusions are as follows: First, that the accommodation reflex is more frequently normal than abnormal; second, that the exaggeration of the reflex is exceptional; third, that loss is more frequent than diminution; fourth, in one-half per cent. it is abnormal upon one side and normal on the other; fifth, exaggeration or diminution are usually moderate; sixth, only during the first period of the disease is the reflex more frequently normal than abnormal, and especially is this the case in the third stage; seventh, exaggeration and unequal changes on both sides were noticed exclusively in the initial stages, while the loss of the reflex was in direct proportion and diminution of the reflex in indirect proportion to the progress of the disease; eighth, an abnormal reflex is noticed in more than one-third of the cases in the period during the remission, and it was only in the demented form that the abnormality of the reflex prevailed over the normal reflex. In all of the other varieties—expansive, depressive, and mixed—the normal was more frequent than the abnormal; ninth, the reflex was more frequently and more profoundly changed in the period of excitement; tenth, changes in the reflex were most frequently noted in the extreme ages—before thirty and after fifty; eleven, exaggeration of the reflex was present, while the motor disturbances were slight. During the first and second periods of the disease the reflex was the more profoundly changed the more the motor disturbances were marked.

Changes in the eye-ground, with progressive amblyopia and amaurosis, may be noted in paresis. However, as compared with tabes, such changes are relatively infrequent. Perhaps, as Klippel points out, this difference is due to the fact that in tabes the peripheral sensory neurone is primarily attacked, while in paresis the peripheral sensory neurone is but rarely and only indirectly attacked,—that is, by the propagation of degeneration from neurone to neurone. The change in the optic nerve, according to Kéravel and Raviart, is more insular in tabes and more annular in paresis. However, lesions of the fundus occur in paresis earlier and more frequently than the older observers thought. That they sometimes occur at a very early period is proven by the three cases placed on record by Knapp. Thus, a man of thirty-seven presented a history of sudden loss of vision in 1889. This loss proved temporary, and vision soon returned. Five months later, however, failure of vision set in, was progressive, and finally became complete. Optic atrophy was present in both eyes. In another case, a man of thirty-six, there was a gradual loss of vision with gray atrophy of both nerves. In a third case, a man of forty-five presented marked impairment of vision of both eyes, while both discs were bluish-gray. In all of these cases the eye-ground changes, according to the author, *preceded* the paresis.

That fundus lesions occur more frequently than was formerly believed is shown by the studies of Kéraval. The latter has made a study of the fundus in paresis and in its initial lesions in fifty-one cases. He found that forty-two cases presented lesions of the fundi. In seven patients in an advanced stage of the disease there were five with white atrophy, one with gray atrophy, and one with sclerochoroiditis. In thirteen cases the papilla was pale. In twenty-two there was involvement only of segments of the papillæ (preliminary stage of optic atrophy). In nine cases in the period of remission there were no fundus lesions. Raviart made a study of the fundus in forty-four cases of paresis. In one there was bilateral white optic atrophy. In ten both papillæ were pale; in two a white papilla on the right side; in four there were gray papillæ; in five the papillæ were pale (or washed out); in six only were the fundi normal. Changes occurred therefore in 73.33 per cent. The majority of the cases in which these changes were observed were in the second stage of the disease. Visual acuity was slightly diminished in the majority of cases.

Individual statistics differ considerably regarding the frequency of optic atrophy. Thus, in contrast to the above results are the findings of Hans Gudden. The latter found primary optic atrophy only sixty-five times in one thousand three hundred and twenty-six cases of paresis. Morselli again, on the other hand, thinks that optic atrophy is very common in paresis. Clifford Allbutt also found forty-one cases of atrophy in fifty-three of paresis, while Tebaldi found the fundus healthy in only one case out of twenty. Boy found optic atrophy in ten per cent., Mendel in five per cent., Joffroy in twelve per cent. Optic atrophy sufficiently marked to cause blindness is relatively rare in paresis. Varying degrees of amblyopia are more common. Finally, optic atrophy, as we have already seen (page 480), may be the first physical sign of paresis.

Disturbances of the visual fields may occur in paresis, but they are not common. This is especially true of marked disturbances. Contracture, however, may be noted, as, for instance, in a case reported by Deutsch of concentric narrowing of the fields. More rarely gross deformities are observed, as in a case of Storch in which, after an epileptiform seizure, a left hemianopsia was noted which persisted for a year.

Among the physical signs of paresis are various palsies of the cranial nerves, more especially of the third, fourth, and the sixth pair. These may give rise variously to strabismus, diplopia and ptosis. They may make their appearance at the very beginning of paresis, or may occur during the period of the fully developed disease. Usually these palsies are not very pronounced or accentuated. Slight degrees of ptosis, weakness of adduction or abduction may be noted, though as a rule careful study is required to detect these anomalies. In this respect they differ markedly from those observed in tabes and syphilis. Quite commonly also they are transitory and fugacious. Dawson, in forty cases, noted palsies of external ocular muscles in only five; Raekes in 18.2 per cent.; Joffroy in 16.7 per cent. Out of his two hundred and twenty-seven cases, Joffroy noted ocular palsies thirty-eight times. In twelve cases a simple ptosis was noted; fourteen times paralysis or paresis of muscles supplied by the third nerve and five times of the muscle supplied by the fourth nerve, five times nystagmus, and twice spasmodic contraction of the orbicularis were noted. On the whole, palsies and other motor disturbances are quite secondary in importance to the other ocular phenomena of paresis; are relatively infrequent and inconstant. At times slight atactic movements and rarely a well marked nystagmus is noted, as witness the cases reported by Deutsch, Hepburn, Ballet, and others. These movements are not to be regarded as truly nystagmic, but merely as a local incoördination comparable to the ataxia and uncertainty of movement noted in the limbs.

SUMMARY.

Among the earliest signs of paresis are inequality and irregularity of the pupil, and at times impairment of the visual acuity and even atrophic changes in the optic nerve. We observe also that the involvement of the visual apparatus is always gradual and progressive. Further we note that it is always asymmetrical, or nearly so.

In keeping with this fact of asymmetry, we find, as the most frequent ocular symptom in paresis, inequality of the pupils. Inasmuch, however, as the inequality of the pupils is very frequent in affections other than paresis, it is obvious that the association of other phenomena is necessary to give to this symptom a specific value. Of itself inequality cannot even serve to differentiate between an organic and a functional disease. As is well known, it may occur, on the one hand, in tabes, paresis, syphilis, or other gross organic disease; or, on the other, in purely functional conditions, such as hysteria and the psychoses. It is, therefore, necessary to search for other signs such as impairment or loss of the light reaction and anomalies of accommodation and convergence. In paresis such additional symptoms sooner or later make their appearance.

Irregularity or deformity of the pupils is almost as common as inequality. Myosis, on the other hand, is less frequent though it occurs, roughly speaking, in about one-third of the cases.

The usual sequence of the phenomena observed is about as follows: at first inequality, irregularity, and impairment or loss of the light reflex, consensual or direct, upon one or both sides, with at the same time preservation of and persistence for a time of the reaction to accommodation; later only do we find impairment and finally loss of the reaction to accommodation, the pupil being then inert to all forms of excitation. The Argyll-Robertson pupil is, therefore, present during a part of the course of paresis. However, we should bear in mind that while this is so in many cases, in others the loss of the light reflex may be somewhat retarded and the loss of accommodation may, on the other hand, occur relatively early, so that the duration of a typical Argyll-Robertson pupil in a given case may be short. In this respect paresis presents a marked contrast to tabes, in which the Argyll-Robertson pupil is a prominent and characteristic feature during almost the entire course of the disease. Observers are not wanting who find the Argyll-Robertson pupil present in but a small number of paretics. Thus, Dawson out of forty cases found it present upon both sides in only five cases, and on one side in only three cases. Intermediate conditions also are found in which the light reflex is not wholly lost, but in which at the same time the reaction to accommodation is reduced. Dawson found this to be the case in 30.76 per cent. Sluggish or slow reaction of the pupil to light with diminished movement is not infrequently observed. It appears to constitute a period between the normal pupil and the typical Argyll-Robertson pupil. Similarly, the accommodation reaction is not infrequently found to be sluggish and slow instead of being lost. Its occurrence means the beginning of a total iridoplegia. Finally we should remember that inequality, irregularity, deformity, myosis, and mydriasis are either forerunners of or are closely associated with anomalies of pupillary action.

The pupillary changes of paresis are, it need hardly be stated, not peculiar to paresis. They are met with in varying degrees in both syphilis and tabes, and some of them, such as inequality, are also found in purely functional nervous and mental affections, as already stated. Not only are all of the pupillary disturbances of paresis observed in tabes, but Babinski and Charpentier have shown that the Argyll-Robertson sign is very frequently met with in old cases of syphilis, irrespective of signs suggestive either of tabes or paresis. Whether the appearance of such symptoms justifies a prognosis of tabes or paresis is still an open question. That it does so, with or without other symptoms and in spite of the presence of the active signs of true nervous syphilis, is an opinion to which the author strongly inclines.

Finally it should be borne in mind that some of the pupillary phenomena observed in paresis *e.g.*, inequality and irregularity—may vary considerably and may even be intermittent; especially may this be noted in the early stages. The light reflex also may in rare instances vary; for example, if greatly impaired though not entirely lost, it may be re-established during a remission as in the case observed by Bumke. It is even asserted that in very rare cases (Fuerstner, Gaup) the reflex to light may be again established after having been entirely lost. On the whole, all of the ocular phenomena of paresis are less fixed and definite than they are in tabes. Again, irregularity of the pupil, so frequently an early sign, is to be looked upon as the beginning of the progressive-iridoplegia of which the Argyll Robertson is a more advanced stage and the inert pupils of the terminal period the final stage.

Eyes which are normal, or at any rate in which no symptom can be detected, are also met with in paresis, but only in the early stages of the affection. Thus, in two cases reported by James, the pupils were equal and reacted properly to light and accommodation. In a third case, however, while the pupils reacted normally, one was smaller in size than the other. In a series of nine cases, studied by Kéraval, eye symptoms were absent in four cases, an experience which must be considered, however, as quite unusual.

Amblyopia, amaurosis, optic atrophy are all physical signs, the detection of which as we have already seen may be of great moment to the diagnosis. That amblyopia, transient amaurosis, and even optic atrophy may be the first noticeable physical sign of paresis should be especially borne in mind.

The extra-ocular palsies and anomalies of movement, though not striking in paresis, should be remembered especially because of their value in differentiating the affection from syphilis, gross organic disease, and perhaps tabes.

The eyes in juvenile paresis present a symptom group similar to that found in older subjects. In keeping with the fact that pupillary changes are apt to be observed at early ages in paresis, Marchand noted sluggish light reaction in a boy of 18 together with inequality of the pupils. Joffroy likewise noted Argyll-Robertson pupils, together with inequality in a juvenile case. Nonne, in a boy of twelve, noted loss of the light reflex in the right pupil and sluggishness of the reflex in the left pupil. The pupils were mydriatic and unequal, while the convergence reflex also was very slight. Raymond, in a girl of twelve, noted diminution of the light reflex, likewise with dilated and unequal pupils. Regis made a similar observation of a patient of twenty-three. There was loss of the light reflex especially upon the left side, together with unequal pupils; there was marked diminution also of the reaction of accommodation. In a boy of eighteen, Marchand also noted sluggish pupillary reactions, the right pupil being larger than the left. Hirschl noted, in a girl of twenty-two, that the right pupil was large and sluggish to light, while the left did not react to accommodation. In a boy of sixteen he noted unequal pupils, loss of the light reflex upon both sides. In a boy of nineteen he again noted that the right pupil was larger than the left and also that there was present a loss of the light reflex. In a man of twenty-four, the same observer noted that the right pupil was larger than the left, with sluggish reaction to light; in the left pupil the light reaction was lost; both pupils presented sluggish reaction to accommodation.

In senile paresis, upon the other hand, Toulouse noted inequality of pupils in a man of seventy-two; Marchand, inequality of the pupils in a man of sixty, and Doutrebente, inequality of pupils in a man of sixty-five, together with sluggishness of the light and consensual reflexes, especially upon the left side.

A subject but little studied and of relatively slight importance is the sympathetic pupillary reaction. This consists of a dilatation of the pupil which ensues upon irritation of sensory nerves of the skin of the face, neck, or other contiguous areas. It is best elicited by pricking or pinching the skin. Hirschl found that in all the cases studied by him, the sympathetic reflex was absent when the Argyll-Robertson pupil was present, except in two. It was also frequently absent when the light reaction was sluggish, and this was also frequently the case even when the light reflex was still preserved. When an Argyll-Robertson pupil was present upon the one side and absent upon the other, the sympathetic reflex was absent upon both sides. The loss of the sympathetic reflex, however, was also noted by Hirschl in senility and alcoholism, and he believes that in cases in which these two factors can be excluded, this symptom offers some diagnostic value. Hirschl also observed that in cases in which this reflex was absent, there were atrophic changes in the sympathetic nerve, sympathetic ganglia, and in the anterior horns of the lower and upper dorsal cord. Moeli found that the reaction was much more frequently lost in cases in which the light reflex had suffered. Hillenberg found similar results.

Among the pupillary phenomena still to be mentioned is that known as the paradoxical pupillary reaction. Here there is an apparent reversal of the light reflex,—that is, an increase of light is followed by dilatation of the pupil and diminution of light by contraction of the pupil. The occurrence of this reaction is an exceedingly rare one in paresis. In fact, but one case, that of Morselli, is known to the writer; in this instance dilatation of the pupil followed the light impression. It is important to bear in mind that a paradoxical pupillary light reaction may be apparent only and not genuine,—for instance, a weakness or paresis of accommodation occurring at the moment that the light test is made, may bring about a dilatation of the pupil. Similarly a paresis of the convergence reaction, accompanied it may be by divergence of the eyeballs, may also occur at the moment that the light test is made, and thus give rise to a dilatation of the pupil. A high degree of amblyopia or amaurosis may be associated with an impairment of accommodation and convergence, and may further account for the occurrence of such a phenomenon. Shock, fright, nervousness, or hysteria may also play a rôle. In practice, as far as paresis is concerned, the paradoxical reaction of the pupil being so rare, may be ignored.

The synergic narrowing of the pupil that accompanies contraction of the orbicularis palpebrarum, and which has been in recent years studied by A. Westphal and Piltz, and which had been earlier noted by von Graefe, by Galassi, and by Moeli, is another pupillary reaction which may be mentioned. It has, however, been studied so little in paresis, that it cannot be said to possess any symptomatic value. It may be added that another pupillary phenomenon, hippus, likewise plays no rôle in the syndrome of paresis.

TABES.

The ocular phenomena of tabes constitute a group of symptoms of great diagnostic value. As in paresis, they may involve varied structures and functions,—*e.g.*, the iris, its movements and reflexes; the various extrinsic muscles, the optic nerve and vision. Tabes is an affection with widely diffused lesions and it is but natural that the early clinical manifestations should vary somewhat in different cases. Accordingly the eye symptoms make their appearance somewhat earlier in some cases than in others.

In general terms, the symptoms of tabes consist of a triad made up of incoördination, loss of tendon reflexes, and the Argyll-Robertson pupil, —the pupil which no longer reacts to light but in which the associated movements of the iris in accommodation and convergence are preserved. The presence of this pupil is not infrequently noted as a very early symptom. It may be noted long before the knee-jerk is lost and before incoordination makes its appearance. Sometimes it antedates all other symptoms by a number of years. On the other hand, its appearance may be much delayed and in rare cases it may not be observed at all even after other symptoms of tabes have existed for many years. While present in the great majority of cases of tabes, it cannot therefore be claimed as a necessary symptom.

It is important in this connection to bear in mind that the loss of the light reflex is a symptom which in all probability makes its appearance gradually. In examining the eye, a difference in the promptness and general character of the light reaction and the reaction to accommodation should always be noted. A striking contrast between the two reactions, such as a slight, slow or sluggish light reflex and a prompt reaction to accommodation possesses an unmistakable significance. As a matter of fact there are many cases in which, in the incipient period, a fully developed Argyll-Robertson pupil is not present and in which a sluggish or other change in the light reaction is the only symptom noted. In a few cases also it has been observed that the Argyll-Robertson pupil is not definitely or permanently established and exists only as an intermittent symptom. Thus Eichhorst reports two cases of intermittent Argyll-Robertson pupils, both occurring in women. Treupel reports another occurring in a man. Camus and Chiray report a most interesting case in which the Argyll-Robertson pupil was present, accompanied by gastric crises. It occurred only during the crises and lasted only as long as the crises.

In estimating the frequency of the Argyll-Robertson pupil in tabes, several factors should be considered. First, while it is true that this pupil is frequently an early symptom, it is not rarely a late symptom; therefore cases of the fully developed disease would, other things equal, present the symptoms in a larger proportion. Secondly, the proportion varies according as the observer excludes or includes pupils in which the light reflex has not been entirely or absolutely lost but in which it has been unmistakably impaired. It is not surprising therefore that there should be some differences in the results of various observers and yet these differences are not as great as one would suppose. Dillmann, for instance, found loss of the light reflex in 76 per cent. of cases in the fully developed stage of the disease. In the early stage he found only 23.7 per cent. Leimbach found the symptom in 75.6 per cent. in the fully developed disease and 47 per cent. in the early stage. The chief variation is found in the determination of the symptom in the early stage and here the element of the personal judgment—the personal equation of the observer—doubtless plays a rôle. On the other hand it is difficult to reconcile the findings of other observers such as Mann, who found the symptom in 79 per cent. of early cases,—i.e., cases in which no pronounced ataxia had yet shown itself. Statistical studies have been made by a large number of observers. Uhthoff, for instance, in three hundred cases noted rigidity of the pupil in 71 per cent.; excluding cases in which a slight reaction of the pupil was still preserved, the percentage became 62. In averaging the results of other observers, for instance, Gowers, Bernhardt, Voigt, Thomsen, Siemerling, Vincent, Dillmann, Berger, Möbius, Marina, Förster and Mann, Uhthoff obtained a percentage of 67, which nearly corresponded to his own results. Among other studies there should be mentioned the following: Harris, 82.6 per cent.; Leimbach, 84 per cent.; Bramwell, 86.1 per cent., and also 72.2 per cent.; Mott, 80 per cent.; H. M. Thomas, 63.6 per cent.; Thoenes, 75 per cent; Collins, 80 per cent.; Fulton, 67 per cent. The writer found among thirty rather advanced cases in the wards of the Philadelphia Hospital 63.3 per cent. These figures do not, it will be admitted, vary very greatly. However, such results as were obtained by F. S. Pearce,—namely, one hundred and fifty cases of Argyll-Robertson pupil in one hundred and fifty-six cases of tabes, that is, 96 per cent.,—are difficult to explain. The percentage found by Riley, who found Argyll-Robertson pupil in only twenty-eight cases out of sixty-one,—namely, 46 per cent.,—must impress one as unusually small. This is also true of Vaughan's results, who found pupillary rigidity in only 40 per cent.

Very interesting and important are the results obtained by Rochon-

Duvigneaud and Jean Heitz, inasmuch as their studies were made by a special method. They examined seventy-seven patients who for the most part were from the service of Professor Dejerine. The patients belonged to all of the periods and the most diverse forms of the disease. The examinations of the pupillary reactions were made in an entirely dark chamber under special precautions in order to determine as far as possible the exact condition of the light reaction. A gas-lamp furnished the illumination. The patient was separated from the latter by an opaque screen. After lowering the gas, without absolutely extinguishing it, the eye to be examined was placed in the most complete darkness for several minutes. Then the operator suddenly raised the gas flame and the light was projected on the eye by means of an ophthalmoscopic mirror. The eye not to be examined remained covered.

Of forty-eight patients examined by these observers, twenty-three had lost all trace of light reflex, fifteen presented a slight reaction to light, and two reacted almost normally but not entirely so. From this it appears that in 47.9 per cent. the light reflex was lost entirely and that in about 35 per cent. it was lost only incompletely; that is, a total of about 83 per cent. presented lost or impaired light reflex, which accords fairly well with the results of other observers. The observations of these writers would go to show that the methods pursued by them reveal a partial preservation of the light reflex when to ordinary tests such preservation would escape notice. Thus they examined twenty-nine cases by the ordinary methods,—*i.e.*, with partial obscurity and moving light. Of these only four presented an incomplete loss of the light reflex, a percentage of 13, as compared with 35. The authors also lay especial stress on the importance of studying the dilatation of the pupil in the dark. This they maintain is an active act, an impairment of which is of profound significance.

Statistical studies for reasons already pointed out can hardly be expected to yield precise results. The conclusion, however, is unquestionably justified, and this indeed is the sum total of individual clinical experience—that the Argyll-Robertson pupil is present in a large majority of cases.

An unilateral Argyll-Robertson pupil is relatively infrequent. Thus, Berger found an unilateral Argyll-Robertson pupil only three times in one hundred and nine tabetics, and Gowers once in seventy-two. H. M. Thomas, on the other hand, states that he found the Argyll-Robertson pupil in seventy out of one hundred and eleven cases and upon both sides in fifty-seven; that is, it was unilateral in thirteen. Probably pupils with but slightly impaired reaction were here excluded. Out of thirty of the writer's cases, it was unilateral in four. It remains an unquestionable fact that in tabes an unilateral Argyll-Robertson pupil is the exception, and there is in this respect a decided departure from the symptomatology of paresis, in which affection this symptom is observed much more frequently.

Finally it should be stated that the Argyll-Robertson pupil may be found when the pupil is of average or normal size, when it is myotic or when it is dilated.

Irregularity or deformity of the pupil is found quite commonly in tabes, as in paresis and syphilis. Joffroy states that he has never seen a case of Argyll-Robertson pupil without some deformity in the latter. This statement, while rather sweeping, accords in the main with the experience of other observers. Deformity is at times the initial pupillary symptom observed and it may be regarded as a precursor of the changes in the pupillary reflexes later to supervene. Joffroy, indeed, regards it as of the same semeiologic value as the Argyll-Robertson pupil. Bumke also insists upon the importance of the loss of the circular shape of the pupil.

Next to the Argyll-Robertson pupil in importance, is the size of the pupil. The pupil may be normal, unusually small, or dilated. Very frequently it is decidedly contracted and this diminution in size may be moderate in degree or excessive. Pin-point pupils are occasionally met with. Myosis frequently accompanies the Argyll-Robertson pupil, though it is by no means a necessary concomitant, for the latter may be associated with pupils of normal size or pupils in a state of mydriasis. Thus, Erb found it in 44 per cent.; Uhthoff, Dillmann, and Voigt found it in a somewhat smaller number. Their findings averaged by Bumke amount to about 24 per cent. Collins found the pupils small in 45 per cent.; the writer in 36.6 per cent. Rochon-Duvigneaud and Heitz found 40 per cent. of bilateral myosis and 9 per cent. of unilateral myosis. In every case of myosis there was a pure Argyll-Robertson pupil, except in three. In the latter contraction to convergence was slow and incomplete. External ophthalmoplegias were not found associated with the myosis. These authors also point out that optic atrophy is more frequently found associated with mydriatic eyes than with myotic eyes. In forty myotic patients, they found only three with total atrophy and blindness and three with beginning atrophy.

Myosis, it would appear, is accompanied quite regularly by the Argyll-Robertson sign. In a number of cases, however, a slight reaction to light is preserved. It is very exceptional, further, to find a myotic patient with optic atrophy and more rarely still with internal or external ophthalmoplegia.

From an analysis of eleven hundred cases, Dufour comes to the conclusion that myosis, associated with other disturbances of the pupils or of the nervous system, confirms the diagnosis of syphilis or organic nervous disease.

Mydriasis is met with much less frequently than myosis. Thus Riley in sixty-one cases found myosis in twenty-eight and mydriasis in eight. Bramwell in one hundred and fifty-five cases of tabes found the pupils contracted in sixty-nine and dilated in eight. In twenty-seven they were of medium size. Collins found mydriasis in only 5 per cent. and when found it was nearly always associated with irregularity of the pupils. Rochon-Duvigneaud and Heitz found mydriasis in 20 per cent. bilateral and 11 per cent. unilateral of their seventy-seven cases. Mydriasis was accompanied by a complicated Argyll-Robertson pupil,—that is to say, a pupil in which the reaction to accommodation was also impaired. These writers point out that when blindness is present, it is as a rule, accompanied by mydriasis. They found seven blind tabetics with bilateral mydriasis and two with unilateral mydriasis. In all of the cases the reflex to convergence was absent or diminished. In twelve cases of mydriasis, they found two with bilateral internal ophthalmoplegia, ten with unilateral ophthalmoplegia, and in two cases there was a nuclear palsy of the third pair, incomplete for the external muscles but complete for the ciliary muscles. In the remaining cases, ten in number, all unilateral, there were five times found besides the internal ophthalmoplegia, also various palsies of the external muscles of the third nerve supply; especially was ptosis found. Once internal ophthalmoplegia with palsy of the sixth nerve was found; four times internal ophthalmoplegia only was present. In five cases of mydriasis there was no blindness or paralysis of accommodation.

The mydriatic pupil is accompanied by various other disturbances, a fact which is directly opposed to the findings in the myotic pupil. Thus the light reflex is totally lost, the convergence reflex is always incomplete or lost, while optic atrophy and ophthalmoplegias are frequent. Finally it is mostly in cases with mydriasis that inequality of the pupils is found.

There is a group of tabetic pupils in which the sphincter iridis presents a reflex paralysis, while the ciliary muscle remains intact and the accommodation is preserved. This paralysis of the sphincter could perhaps be attributed to a limited nuclear lesion, for the nucleus of the circular fibres of the iris is entirely distinct from that of the ciliary muscle. Again, while paralysis of accommodation is often accompanied by paralysis of the external muscles, in not one case did Rochon-Duvigneaud and Heitz find such a paralysis associated with the isolated palsy of the sphincter iridis.

It has been observed that during a gastric crisis, pupils previously noted as normal may dilate, but that in such case they return to their former size after the crisis has subsided. Such an observation has been made by Rochon-Duvigneaud and Heitz, dilatation and subsequent return to normal occurring in one of their patients at every gastric crisis. Similar observations have been made by Trousseau and Raymond.

Schmeichler states that he has never seen unusually large pupils in the early stages of tabes. This is an important observation and in keeping with general experience.

It is interesting to note also that atropine gives an incomplete or imperfect dilatation of myotic pupils, and that pilocarpine in mydriasis yields a contraction which is not very pronounced.

Mydriasis, according to Rochon-Duvigneaud and Heitz, always accompanies the complicated Argyll-Robertson pupil,—that is, the pupil in which the light reaction is totally abolished, but in which the reaction to convergence is also lost or is poor. In a certain number of cases, mydriasis depends upon blindness. When the mydriasis is accompanied by a preservation of vision the state of the accommodation should be investigated, taking into account, of course, the age and refraction of the patient. Usually under these circumstances the accommodation is paralyzed. There is then a total internal ophthalmoplegia, involving the sphincter iridis and the ciliary muscle. Very rarely temporary dilatation of the pupils, that is "alternating mydriasis," is met with. It is much less frequent in tabes than in general paralysis.

Inequality of the pupils should, even if slight, be regarded with suspicion, especially if one of the pupils reacts sluggishly to light or shows impairment of the light reflex, no matter how slight. Collins noted inequality in 23 per cent. of his cases. He adds that it was probably not noted in cases in which the inequality was very slight. Bramwell found the pupils unequal in 34.8 per cent. Vaughan found 60 per cent. of inequality of the pupils, a percentage which it cannot be denied is out of keeping with general experience. The writer found 46.6 per cent. of decided inequality and 13.3 per cent. of very slight inequality. It will be noted that the percentage of inequality in tabes is decidedly less than in paresis. Among other statistics are those grouped by Bumke,—namely, Bernhardt, who saw inequality of the pupils in 43 per cent., Voigt in 40 per cent., Dillmann in 34 per cent., Förster in 33 per cent., Uhthoff in 28 per cent., and Berger in 27 per cent. Inequality, if observed as an isolated sign and without other ocular or nervous symptoms, has no special significance. In other words, inequality may be perfectly physiological. It must, however, be borne in mind that a physiological inequality is usually slight. (See Chapter VI, p. 314).

The optic nerve is frequently involved in tabes. There is present in such cases a simple and progressive atrophy of both optic nerves,—an atrophy which may and frequently does lead to complete blindness. It is a symptom which is frequently associated with an Argyll-Robertson pupil, but it may exist without the latter and it may be the first and only symptom. Indeed, simple optic atrophy, in the absence of other signs, is very suggestive of tabes. Usually it makes its appearance early; it is only in exceptional cases that optic atrophy is a late symptom. Collins believes that the discoloration of the optic nerve begins within five years after the onset of the disease. He regards it as an early symptom and believes that the changes probably start peripherally in the retina. So frequently is early optic atrophy noted that it is justly counted along with the Argyll-Robertson pupil as one of the first symptoms of tabes. Mott also regards limitation of the visual field and optic atrophy as the early signs of tabes. Gowers has seen optic atrophy fifteen or twenty years before other tabetic signs made their appearance. Not infrequently one eye is involved earlier and to a larger extent than its fellow, but the optic atrophy is never really unilateral. Defective vision is the eventual result. The first indication is a narrowing of the visual field. The impairment usually consists of an irregular concentric contraction; at times the field is irregularly indented. There may be little or no diminution of the acuity of the central vision and no visible pallor of the disc. Central scotomata are very exceptional; when present they are frequently explicable on the ground of a complicating alcoholism. Occasionally the visual field is limited on one side, as, for instance, in the case reported by Halbau, in which the visual field in the right eye, especially for red, was limited on the temporal side. Hemianopsia, incomplete in character, is a rare anomaly of the field and is apparently due to an asymmetrical degeneration. It has been noted by Gowers, Berger and Gabrielides. In Gowers's and Gabrielides's cases, there was a double temporal hemianopsia referable to a change in the chiasm. In Berger's case, there was a right homonymous hemianopsia. There were also present Argyll-Robertson pupil, double optic atrophy, palsy of the right fourth nerve and other symptoms of tabes. The loss may also involve early the color perception, the vision for green, red, and blue being affected in the order named. Dyschromatopsia was first noted in tabes by Benedict. Generally speaking dyschromatopsia is partial and concerns mostly red and green, the red being taken for black and the green for gray. A curious symptom occasionally noted in tabes is green vision. Such a case, for instance, has been placed upon record by W. Dodd, in which the patient saw everything a bright emerald green in color. Occasionally he saw rose-colored spots in the green. These colors increased in intensity when he was fatigued. He occasionally also saw a brilliant light. In 1891 diplopia had been noted from which the patient recovered. Ocular vision remained good until 1897. Examined in September, 1898, green vision was noted. The vision of the right eye was 2/60, of the left 1/60.

Rarely optic atrophy is accompanied by photophobia and blepharospasm.

Statistics as to the relative frequency of optic atrophy in tabes vary very widely and are on the whole unsatisfactory. Collins found optic atrophy in 14 per cent., Bonar 8 per cent., Thomas 10 per cent., Riley 11.4 per cent., Bramwell 22 per cent., Sarbo 30.4 per cent. Fulton found only 2 per cent., Vaughan noted 50 per cent., Dillmann 42 per cent., Berger 44 per cent., Leimbach 16.75 per cent., and Grosz in 88 per cent. Mott places the percentage of cases of optic atrophy in tabes at 80. Pearse, on the other hand, found only thirty cases of optic atrophy in one hundred and nineteen cases. Marina observed optic atrophy in 9 per cent., Grosz in 88 per cent., Uhthoff in 20 per cent., Bernhardt in 10 per cent., Berger in 44 per cent., and Silex 15 per cent. Oliver believes that optic atrophy is met with in 10 to 15 per cent.

It has long been held that when tabes begins with optic atrophy that the affection soon becomes arrested. The more exhaustive investigations, however, of André Léri fail to substantiate this position. Numerous observations fail to prove that the onset of optic atrophy is attended by a cessation of the progress of the disease. A case reported by Bramwell will serve as an illustration. In a man of thirty, two years previous to all signs of tabes, optic atrophy with blindness and dilatation of the pupils were present. Six years after, however, other important signs of tabes made their appearance. Collins also noted that in no case did the supervention of blindness seem to have any effect upon the course of the disease. Gowers maintains that optic atrophy is generally an early symptom, beginning before incoördination is developed and that in a large number of cases ataxia does not supervene. Benedict believes that tabetic motor symptoms cease after they have attained a high degree, or gradually after the optic atrophy appears. In eight out of thirty-three cases observed by Bramwell, the development of the optic atrophy seemed to be attended by an amelioration of the symptoms and a retardation of the tabetic process. In twelve cases, however, the development of the optic atrophy was not followed by an amelioration of the symptoms and had no retarding effect upon the course of the disease. In nine out of twenty-six cases in which optic atrophy was very marked, the development of the optic atrophy coincided with a rather apparent arrest in the tabetic process.

The details obtained by some observers are interesting; thus Bramwell, who studied one hundred and fifty-five cases of tabes, noted that in seven of his cases, in which optic atrophy was present, the symptom was much more marked in one eye than in the other and that in three cases it was limited to one eye. In seven cases it was slight, in seven marked, and in twenty-one very marked or complete, the patient being almost blind, quite blind, or only able to distinguish light from darkness. In one case, double optic neuritis, marked in the left eye and slight in the right, preceded the optic atrophy. Such an occurrence suggests, however, an active syphilitic process rather than tabes. In nine patients dimness of vision, due to optic atrophy, was the initial symptom of the disease. In twenty-five of the cases in which optic atrophy was present, there was ataxia.

Ocular palsies are extremely common in tabes and in quite a large number of cases they occur as very early symptoms. They involve variously the third, fourth and sixth nerves. They usually appear suddenly and may disappear with or without treatment. Their duration is usually short, lasting a few weeks, a few days, or indeed only a few hours. They may, however, be permanent, though this is rare and points to a syphilitic rather than to a tabetic origin. However, a persistent ocular palsy may be present in the very beginning of the affection but is more frequently observed in the later stages of the disease.

The ocular palsies of tabes present nothing that is characteristic. Though they develop rapidly, they usually disappear gradually and sometimes slowly. The muscular involvement is seldom complete and is usually limited to one eye. Further, the palsy is never a palsy of associated muscles or movements, but always involves, irregularly, different muscles of one or both eyes. Occasionally a complete ophthalmoplegia appears. This again is to be regarded as pointing to a syphilitic rather than a tabetic origin. It is noteworthy that in syphilitic ocular palsy, a large number of muscles are affected at the same time while in tabes involvement of a single muscle is the rule. It is further interesting and in practice important to note that it is extremely rare in tabes to find ocular palsies without a loss of the light reaction. Opinions differ as to the muscles most frequently involved. Thus Swanzy and others believe that the external rectus is most frequently affected, while Charcot believed that the oculomotor nerve supply suffered first. Javal found that the levator was most frequently involved, then the internus, the externus and the superior oblique. Certain it is that ptosis is very frequent. It may be slight or pronounced and may be single or double. It does not always depend upon paralysis, especially when slight; it is then due to atony of the levator and under such circumstances may be inconstant. Finally it rarely exists alone. Very frequently other muscles are involved, giving rise to a diplopia. This diplopia is usually due to a palsy in one eye, is very common, and is usually transitory. Transitory diplopia is frequently an important point in the history of the early period of a case of tabes. At times the diplopia depends upon palsy of the external rectus, at times it depends upon palsy of the internus or other muscles of the oculomotor supply,—*e.g.*, the superior rectus. Involvement of the fourth nerve,—especially isolated involvement,—is infrequent, if not rare. The diplopia, though transitory for the most part, is in rare cases permanent. Further, having once disappeared, it may recur. It is sometimes an exceedingly early symptom; thus, in a case reported by Harris, it existed for six years before other symptoms of tabes made their appearance.

Rarely muscles other than the ocular muscles may be palsied at the same time. Thus in a case reported by Turner, a man of forty, there was some ptosis and also some bilateral palsy of the soft palate and paralysis of the vocal cords. Rarely also instead of an ocular palsy, a blepharospasm may be noted or a blepharospasm may precede the development of ocular palsies.

A most interesting form of ocular palsy has been reported by Egger. In one of his clinics, Dejerine presented a case of bulbar tabes in which of the twelve pairs of cranial nerves, the affection had only spared the IV pair, the III, V, VI, VII, VIII, IX, X, XI, and XII pairs were all more or less strongly involved. A bilateral and absolute deafness characterized the lesion of the VIII pair, whilst a slight ptosis of the left upper eyelid and a paresis of the left internal rectus were the sole manifestations of involvement of the III pair. Although there was no paralysis of any one muscle of the eye, there was notwithstanding a marked reduction in the motility of the eyes. When in the normal individual the head is turned to one side—for example, to the left on its vertical axis—the eyes in order to preserve their primary position move slowly in the opposite direction—*i.e.*, to the right—and compensate by movement equivalent to the degree of rotation effected by the head, but at a given moment the eyes abandon this primary position and a return movement ensues. This go and come repeats itself about a dozen times for one complete rotation of the body around its vertical axis and gives, if the rotatory movements acquire a certain speed, the aspect of a nystagmus which has been called the transverse nystagmus of rotation. These movements are regulated by the pressure of the liquid in the semicircular canals, varying for each position of the head. Each canal is sensitive for the movements affecting its plane. A rotation in a horizontal plane to the left, however, affects only the left horizontal canal, in the interior of which the liquid, thanks to the law of inertia of matter, concentrates toward the ampulla and there produces an excitation which informs the centre as to the direction of the change of position. The movements in the sagittal or vertical plane are only registered by the sagittal or superior-semicircular canals and the movements in the frontal and transverse planes by their respective canals, and as each canal perceives only one movement—that is to say, the change of position in its own plane—it can only excite a compensatory movement which the direction of the change of position calls forth. It is thus that the horizontal canals preside over the compensations of lateral movements in innervating the external and internal recti. The sagittal canals regulate the vertical oscillations and the frontal canals the muscular apparatus affecting compensatory rotation around the visual axis. If the tips of the middle and ring fingers of each hand are lightly placed against the eyeballs in the healthy man, these movements can readily be felt. In Egger's case not the least trace of movement was transmitted to the fingers for any change of position. The phenomena of nystagmus of rotation and of post-rotatory nystagmus were lost. In semidarkness, where the retinal reflex did not impede by the fixation of objects the influence of the labyrinth, he observed in the two open eyes that the latter, instead of making a movement opposite to that of the direction of the head in order to preserve their primary position, remained absolutely immobile in their orbits. They did not participate in the movements of the head. This absence of compensatory movement is the rule with deaf-mutes and is observed in some cases of internal otitis. Among tabetics it had up to the case reported by Egger not been observed. The name labyrinthic ophthalmoplegia has been proposed by Egger for this symptom.

The cause of the diplopia or ptosis of tabes appears, according to Harris, to be some vascular derangement or slight ependymitis in the nuclear region. Pierret believes that it begins as a nuclear affection and extends gradually downward toward the periphery as a descending neuritis and here continues after the nuclear involvement has disappeared. Dejerine says that not all ocular palsies in tabes are of nuclear origin, and Swanzy claims that a complete and permanent paralysis of an isolated muscle is due to a peripheral lesion and not nuclear. Complete oculomotor palsy, rarely noted as an early symptom, is probably always due in part at least to an involvement of the oculomotor nucleus. This was the finding in a case of Marina in which a third-nerve palsy disclosed at autopsy a degeneration of the oculomotor nucleus besides a neuritis of the trunk. It is probable also that in cases where an ocular palsy becomes permanent, there is also involvement of the nucleus.

Some idea of the frequency of ocular palsies may be gained from the following figures: Thus, H. M. Thomas found affections of the external muscles in thirty-three out of one hundred and eleven cases. G. Oram Ring states that palsies of the ocular muscles occur in from twenty to twenty-five per cent., and that they are usually found in the preataxic stage and not infrequently exist as initial symptoms. Harris noted diplopia and other ocular palsies in 11.3 per cent. of his cases. Diplopia due to paralysis of the external rectus of one side, Riley found in fourteen of sixty-one cases. Bramwell noted some form of ocular palsy in 16.1 per cent., ptosis in 7 per cent. In seven of his cases, the ptosis was unilateral, in four bilateral. Paralysis of the external rectus occurred in 6.4 per cent., unilateral in six, bilateral in four. More extensive paralysis of the external muscles occurred in 2.5 per cent. Leyden and Goldscheider found palsy of external ocular muscles in from 40 to 50 per cent. of their cases. (See Chapter V, p. 209).

Next, perhaps, in clinical importance to the phenomena thus far considered are loss or impairment of the reactions of the pupil to accommodation and to convergence. Loss or impairment of these functions may exist together with loss of the light reflex, though such an association is infrequent. Further, when accommodation and convergence are lost, it is usually in connection with a large and dilated pupil. Ophthalmoplegia interna is very rare in tabes and, as may be inferred, is indicative of an active syphilitic process rather than tabes. Uhthoff, however, found ophthalmoplegia interna in 5 per cent. of his cases and, it should be added, always unilateral. The latter fact alone suggests the special character of the lesion at work, a lesion not common to tabetics as a whole.

It appears that paralysis of convergence is somewhat more rare than paralysis of accommodation. Thus of thirty cases studied by the writer, paralysis of accommodation was found twice, though six cases revealed an impairment of accommodation. The convergence reaction was absent but once and impaired but twice. Similar results were obtained by Rochon-Duvigneaud and Heitz. These investigators found only three cases with loss of convergence in seventy-seven. Collins again found in one hundred and forty cases loss of accommodation in nine.

Paradoxical pupil is an exceedingly rare symptom in tabes, if indeed it ever really occurs. In such a pupil, dilatation follows exposure to light, while contraction ensues upon darkness. This phenomenon is open, however, to gross misinterpretation. Very frequently the dilatation observed is in reality due to an impairment of accommodation, or more rarely to a weakness of the convergence reaction. The sphincter of the iris being weak makes, perhaps, an ineffectual attempt to shut down and then suddenly gives way and dilates. In the only case ever observed by the writer, involvement of accommodation and convergence was clearly present. Piltz (cited by Bumke) reports a case of double optic atrophy (tabetic?), in which upon sudden shading of one eye, both pupils became distinctly narrower. Upon sudden illumination, the left pupil did not react at all, while the right contracted slightly, both directly and consensually. It would appear that the literature fails to offer a case entirely free from objection. (See also page 493.)

Hippus, though very rare in paresis, has been noted in tabes, as, for example, in the case noted by Hartman. The case was that of a girl of twenty. The pupils were otherwise normal and with normal reactions. There was present, however, a high degree of optic atrophy with marked impairment of vision and concentric contraction of the visual fields.

Atactic movements of the eyeballs are not infrequently observed, though they rarely approximate a true nystagmus. It is probable that this symptom when present in the lesser degree is expressive merely of the general factor of incoördination, though when true nystagmus is present a special cause seems indicated. Harris suggests that it is probably due to nuclear disease. Bramwell noted jerking on extreme lateral deviation in 2.5 per cent. of his cases. Fulton noted it in 2 per cent. of his cases. In two out of thirty cases examined by the writer nystagmus was present; in two atactic movements, moderate in degree, and in eleven very faint atactic movements were noted.

Pain in or about the eyeball is sometimes observed, especially early in the history. It not infrequently assumes the form of a periorbital neuralgia. Such a neuralgia may prove a precursor of optic atrophy, as in a case observed by Galezowski. Areas of anæsthesia may be found also in the course of the fifth nerve. Very rarely the pain assumes the form of ocular crises, as in the case reported by Pel. In the latter the patient suddenly experienced violent burning and knife-like pain in and about both eyes. There were present also cramp-like contractions of both orbiculares, intense lachrymation and redness and injection of the conjunctivæ. The neighboring structures were hyperæsthetic. The crises lasted from two to three hours. They were attributed by Pel to a neuritis of the ciliary nerves. They call to mind very forcibly the attacks termed ophthalmic migraine every now and then noted in paresis. (See Chapter XVII.)

Lachrymation is not infrequently observed in tabes and is regarded by some writers as an early symptom. It is not infrequently associated with myosis. Bramwell found it in 1.2 per cent. of his cases.

Other pupillary phenomena than those mentioned above may be observed in tabetics, but they have very little clinical value. Such, for instance, is the psychic pupillary reflex of Piltz which is independent of the presence of a direct light, the patient's pupil reacting or contracting in accordance to his mental conception of the presence of a bright light or darkness. Similarly in blind tabetics, one can occasionally secure movements of the pupil by asking the patient to fasten his mind upon some object supposed to be in the distance and then again upon some object which is close at hand. In such case a psychic reaction to accommodation may be observed. The writer has demonstrated such a reflex in the case of a blind tabetic sailor, who, when asked to think of a ship barely discernible upon the horizon, would have a dilatation of both pupils, and when asked to think of reading a newspaper in his hand, the pupils would distinctly contract. The synergic pupillary reaction of Piltz and Westphal may also be observed in tabetics, though it has no special clinical value. Piltz found that the pupil contracted synergically with the orbicularis palpebrarum in 43 per cent. of tabetics. The writer found it present in only 3 per cent. of his cases and in most of these it was slight and not pronounced. In one of the writer's cases the pupil, instead of contracting, distinctly dilated.

For the most part also, the reflex dilatation of the pupils to sensory irritation is found to be absent in tabetics. It was actually present in but two of the writer's cases; faint dilatation was noted in a slightly larger number.

SUMMARY.

The ocular findings of tabes consist briefly of the Argyll-Robertson pupil, of atrophy of the optic nerve and of ocular palsies, the latter being usually fugacious and rarely persistent. Any or all of these symptoms may make their appearance early and not infrequently antedate the general symptoms of tabes. Impairment of the light reflex and beginning optic atrophy vie with each other as to the frequency of their early appearance. Ocular palsies also, as we have seen, may make their appearance at an early stage. Again, irregularity of the pupils may antedate a distinct impairment of the light reflex and its occurrence should always be regarded with suspicion. The same is true of inequality, though this is not observed as often in tabes as in paresis.

Myosis, other things equal, is also an early symptom; mydriasis, on the other hand, is a relatively late symptom and is frequently associated with advanced optic nerve atrophy and blindness. At times it is also associated with loss of the reaction to accommodation and of the reaction to convergence; ophthalmoplegias also are frequently found associated with a mydriatic pupil. Finally, loss of the accommodation and convergence reactions, if present, are usually observed in a rather advanced stage of the disease. An initial ophthalmoplegia at an early stage, it will be remembered, speaks rather for a syphilitic lesion than for tabes.

The eye symptoms of juvenile tabes on the whole resemble those of the disease in the adult. An analysis of twenty-four cases, reported by various authors, shows that an Argyll-Robertson pupil and beginning optic atrophy are among the early symptoms. Isolated muscle palsies, strabismus and ptosis are also present. In a case reported by Bloch there was inequality of the pupils, the left being myotic. The right pupil did not respond either to light or accommodation, the left reacted to light. In a case reported by Janus, a woman of twenty, the upper eyelids were slightly retracted, the left more than the right, the pupils unequal, the right larger than the left, both Argyll-Robertson and complete paralysis of the external rectus. In another case, a boy of seventeen, there were gross choroido-retinal changes in the right eye, together with vitreous opacities. The right pupil was sluggish to direct light but active consensually. In another case, a girl of fifteen, the changes were limited to unequal pupils, the right being larger than the left; both reacted feebly to light. In four out of six cases in infantile tabes, collected by Idelsohn, the Argyll-Robertson pupil was present. In four cases of juvenile tabes, reported by Maas, there was in one gray optic atrophy and Argyll-Robertson pupils in both eyes. In another unequal pupils, loss of the reflex to light and convergence in both eyes and also choroido-retinitis. In two others there were present unequal pupils, gray atrophy and Argyll-Robertson pupils upon both sides. In one of these there was a coloboma of the left eye.

FUNCTIONAL INSANITIES.

In the insanities,—that is, the functional psychoses (excluding, of course, under this head paresis, tabes with psychoses and organic brain disease generally),—the ocular symptoms presented are relatively unimportant and possess but little clinical value. There are no pupillary changes which are peculiar to any mental disease. All efforts that have aimed to bring special ocular symptoms in accord with individual insanities have failed. Notwithstanding it is of importance to review briefly some of the studies that have been made in this field and to consider briefly the inference that it is possible to draw from them.

The changes that have been studied relate mainly to the pupil.

According to Dagonet, disturbances of the motility of the iris and inequality of the pupils may be found in both normal and insane individuals. In the latter it is in the chronic forms that anomalies are more frequently observed. In mania a dilatation was observed by Dagonet, a dilatation which may alternate with exaggerated contraction of the pupils. Minck found spasmodic myosis in periodic mania and in the puerperal psychoses, and mydriasis in acute mania. Regis writes, pupillary inequality with variable light and accommodation reflexes is often observed in psychoses due to autointoxications, but this inequality is very changeable from one day to another and often on the same day. Stephani and Morpujo see a relation between the degree of myosis and development of the psychic disturbances. Thomsen says that pupillary immobility exists in 2.1 per cent. of the insane, exclusive, of course, of paresis. He found it once among one hundred and eighty-nine epileptics, in four out of two hundred and ninety-eight patients with systematized delusions, and in fourteen out of six hundred and seventy cases of other forms of insanity. In 1885, Siemerling examined seven hundred insane women. He found an absence of the light reflex in three cases of senile dementia, in one of syphilitic dementia, in three tabetics with psychoses, in two epileptics, and in one case of systematized insanity; in all the other cases the light reflex was unaffected. In 1896 he published a statistical study concerning nine thousand one hundred and sixty cases. He noted the absence of the light reflex in twenty-nine cases of tabes with psychoses, in seventeen cases of cerebral syphilis, in fifteen cases of alcoholic dementia, in nineteen cases of senile dementia, seven cases of paranoia, in four cases of hysteria, and in four cases of epilepsy. The above facts certainly justify the inference that disturbance of the light reflex is very rare among the insane.

Moeli examined nineteen hundred patients. He found immobility in fifty-six, but excluding organic cases, such as paresis, the percentage was only 0.8. Oebeker noted inequality in 2 per cent. Mignot says that, generally speaking, the percentage will be 2, and, since the difference between paresis and other forms is so great, one can safely consider pupillary disturbances as belonging to paresis more than to any other form of mental disease. According to Verga and Castigliani, no changes of pupils are pathognomonic of insanity.

Mignot analyzed one hundred and forty-four cases; paresis was not included. He found inequality in 39 per cent., disturbed light reflex in 43 per cent., disturbed accommodation in 69 per cent., deformities in 38 per cent., myosis in 17 per cent., mydriasis in 35 per cent. These results are analogous to the observations of Verga and Castigliani.

In psychoses following *organic* lesions of the brain, Mignot found marked pupillary changes in all. Thus in a case of maniacal excitement, following hemiplegia, the left pupil was larger than the right, the light reflex was absent and the pupils were deformed. In a case of melancholia with mental debility after hemiplegia in a diabetic, the right pupil was larger than the left and deformities were present. In a man suffering from paranoia, but also presenting a history of cerebrospinal syphilis, the right pupil was larger than the left, there were loss of the reflexes to light and accommodation and deformities of both pupils. In a fourth case, a woman suffering from dementia following cerebral syphilis, the pupils were 5 millimetres in diameter, presenting incomplete light reflex and no convergence reaction.

In senile dementia, Mignot found pupillary changes in all of the five cases examined by him. Thus one, a man of eighty, presented a myosis of 1 millimetre with absence of the reflexes to both light and accommodation. A second, a man of eighty-seven, presented pupils of 2.5 millimetres with very sluggish reaction to light, no accommodation reaction, and deformities. In a third, a man of seventy-eight, presented pupils of 2 millimetres with loss of both light and accommodation reactions. A fourth, a woman of eighty-one, presented pupils of 3 millimetres with likewise loss of light and accommodation reactions. A fifth case, a man of seventy-three, presented pupils of 5 millimetres with impaired light and accommodation reactions. Mignot also studied the pupillary changes in the precocious dementias in sixty-three cases. Of these sixty-three cases, nineteen had dilatation of the pupils; nineteen presented inequality. The light reflex was examined in fifty-three cases; four times—*i.e.*, in 7.54 per cent.—it was found lost and in twenty-nine—*i.e.*, 54.71 per cent.—it was found altered. In the twenty-nine were included twelve cases in which variations of the light reflex were noticed,—*i.e.*, at certain times it was absent, at other times diminished, at other times normal. This variability appears to Mignot to be characteristic of the pupillary disturbances in dementia præcox. In thirty-nine cases examined for the convergence reflex five presented disturbances. Finally thirty-one out of fifty-nine cases showed deformity. In detail the results are as follows: Inequality, 32.20 per cent.; disturbed light reflex 62.26 per cent., disturbed accommodation reflex 12.81 per cent., dilatation 30.15 per cent., and deformities 52.64 per cent.

Marandon de Montyel's investigations show that abnormality of pupils occurs far more frequently in the chronic insane than in the acute. In twenty acute cases it was normal in 50 per cent. and abnormal in 50 per cent. In fifty-seven chronic cases it was normal in 5.2 per cent. and abnormal in 94.8 per cent. He characterizes as chronic cases those that have a duration of two years at least of confirmed insanity. Even after this long lapse of time it is not extremely rare for recovery to follow. Paresis is doubtful only in the initial phase of the disease; after a duration of two years the symptoms are so pronounced as to leave no doubt as to the diagnosis. Therefore this rule can be of little avail for the differentiation of paresis. One out of two cases of acute insanity has an abnormal pupil. Disturbances and deformities were noted in the acute and chronic cases in the following percentages. Thus, in the acute, disturbances were noted in 15 per cent.; deformities in 20 per cent. and disturbances and deformities together in 15 per cent. In the chronic cases, disturbances were noted in 50.8 per cent., deformities in 8.7 per cent. and disturbances and deformities together in 35 per cent. The disturbances are nearly three times more frequent in the acute than in the chronic cases. In detail the special symptoms were found in the following proportions:

	Acute. Per cent.	Chronic. Per cent.
Inequality	3 (15)	26 (45.6)
Myosis	0 (0)	10 (17.5)
Mydriasis	0 (0)	12 (21)
Deformities	7 (35)	25 (43.8)
Alterations of light reflex	2 (10)	32 (56.1)
Alteration of accommodation reflex	1 (5)	20 (35)
Bilateral identical alteration	0 (0)	15 (26.3)
Argyll-Robertson pupil	2 (10)	19 (33.3)
Inverse sign	1 (5)	6 (10.5)
Paradoxical reaction	0 (0)	1 (1.7)
Hippus	1 (5)	2 (3.5)

Regarding the involvement of one or both eyes, de Montyel found that the deformities were unilateral in four of his acute cases and nine of the chronic. In three acute cases and in sixteen chronic cases the deformities were bilateral. As regards the inequality of the pupils, he found a difference of less than a half millimetre in two acute and eighteen chronic, from one-half to one millimetre in none of the acute and six chronic, one millimetre and more in one acute and two chronic.

In regard to deformities, de Montyel found them present upon one side in four acute and eight chronic cases; upon the two sides in two acute and four chronic cases; two deformities in one eye in none of the acute and one of the chronic cases; two deformities in each eye in none of the acute and none of the chronic cases; one deformity in one eye and two in the other in one of the acute and two of the chronic cases.

These facts prove that one may find in the acute insanities deformities of the pupils as pronounced as in the chronic insanities. De Montyel concluded also that the maximum deformities were more frequent in the acute than in the chronic cases. The acute cases would furnish a deformed pupil in the proportion of one in seven and the chronic one in more than a dozen. De Montyel further analyzed fifteen of his acute and fifty of his chronic cases to show the evolution of the pupillary changes with the following result: He found the pupils normal throughout in 40.1 per cent. of the acute cases and in only 4 per cent. of the chronic cases. He found them abnormal throughout in 33.3 per cent. of his acute cases and in 90 per cent. of his chronic cases. It was first normal and later abnormal in 26.6 per cent. of his acute cases and in only 6 per cent. of his chronic cases. It was abnormal and later normal in none of the acute cases and in none of the chronic cases. According to this result, 40 per cent. of the acute cases have normal pupils as long as they are in the acute stage, while only 4 per cent. have normal pupils in the chronic stage. Therefore 36 per cent. of normal pupils of acute cases will become altered should these cases become chronic. In none of the cases, acute or chronic, examined by the author did he see pupils, once changed, again become normal, while in over one-fourth of the cases pupils normal in the beginning presented changes later.

Marandon de Montyel also made a study of the pupillary disturbances in summer and winter and he analyzed seventy-four in summer and sixty-nine in the winter, a large proportion of the latter being cases studied by him in the preceding summer. In the summer he found 25.7 per cent. normal and 74.3 per cent. abnormal. In the winter he found 15.9 per cent. normal and 84.1 per cent. abnormal. The abnormal changes showed the following: Changes were present in 43.2

per cent. in the summer and in 42 per cent. in the winter. Deformities were present in 12.1 per cent. in the summer and 13 per cent. in the winter. Changes and deformities together were noted in 16.2 per cent. in the summer and in 29 per cent. in the winter,—that is, there was a noticeable increase in keeping with the duration and the tendency to chronicity.

De Montyel's conclusions are as follows: In the majority of cases the pupils are altered in form and function in the insane; while the changes are mostly slight, there is nevertheless a large proportion in which they are marked. They are almost always persistent and even increase in intensity. These facts de Montyel considers of great importance for the differential diagnosis of paresis.

Marandon de Montyel has studied the pupillary disturbances and deformities in the various forms of insanity. He makes an analysis of seventy-seven cases. The pupils were normal in 20 per cent. of mania, in 10 per cent. of melancholia and in 0 per cent. of dementia, in 0 per cent. of circular insanity and in 100 per cent. of insanity with systematized delusions. They were abnormal in 80 per cent. of mania, in 90 per cent. of melancholia, 100 per cent. of dementia, in 100 per cent. of circular insanity and in 0 per cent. of insanity with systematized delusions. The abnormalities noted were as follows:

	Mania	Melancholia	Dementia	Circular
	Per cent.	Per cent.	Per cent.	Per cent.
Inequality	6 (40)	15 (37.5)	6 (54.4)	2 (40)
Myosis	0	8 (20)	2 (18.1)	0
Mydriasis	0	10 (25)	1 (9)	1 (20)
Deformities	7 (46.6)	17 (42.5)	6 (54)	2 (40)
Changed light reflex	5 (33.3)	17 (42.5)	8 (72.7)	4 (80)
Changed accommodation reflex	6 (40)	10 (25)	4 (36.3)	1 (20)
Argyll-Robertson	4 (26)	8 (20)	6 (54.4)	3 (60)
Reversed sign	3 (20)	2 (5)	2 (18.1)	0
Paradoxical pupil	0	1 (2.5)	0	0
Hippus	1 (6.5)	2 (5)	0	0

The unilateral or bilateral character of the symptoms is indicated in the table on the next page.

The general conclusion can be drawn from it that bilateral changes predominate. All symptoms which show some tendency to inequality and deformities are to be noted.

As to the method of involvement of the special reflexes of light and accommodation, de Montyel achieved the following results: In mania the reflex was at no time exaggerated, in melancholia he found it exaggerated twice, in dementia never, never in circular insanity. He found it diminished three times in mania, eleven times in melancholia, three times in dementia and three times in circular insanity. It was much diminished in one case of mania, in one case of melancholia, in four of dementia, and in none of circular insanity. It was lost in one case of mania, three of melancholia, one of dementia, and one of circular insanity.

	Mania		Melancholia		Dementia		Circular	
	Unilateral	Double	Unilateral	Double	Unilateral	Double	Unilateral	Double
Myosis	0	0	2	6	0	2	0	0
Mydriasis	0	0	8	7	0	1	0	1
Deformities	8	4	7	10	1	5	2	0
Light reflex	0	5	0	17	0	8	0	4
Accommodation	0	6	0	10	0	4	0	1
Paradoxical	0	0	0	1	0	0	0	0
Hippus	0	1	0	2	0	0	0	0

The accommodation reflex was not found exaggerated at any time in mania, melancholia, dementia, or circular insanity. It was diminished in four cases of mania, four of melancholia, four cases of dementia and one of circular insanity. It was much diminished in one case of mania, two of melancholia, in none of dementia and in none of circular insanity. It was lost in one case of mania, and in four of melancholia, in none of dementia and in none of circular insanity.

De Montyel's conclusions are as follows: In systematized forms of insanity the pupils are constantly normal.

In dementia the pupils are constantly abnormal. Identical changes are present in both reflexes to light and accommodation, while deformities are present in general and are multiple; a number of disturbances are present in the same patient and at the same time. There is a marked diminution of the light reflex. Deformities alone are rare. Paradoxical reaction is not found and there is no hippus.

In circular insanity there is a constant abnormality of the pupils. There is a maximum of association of deformities and disturbances in the same individual. There is a tendency to unilaterality of deformities which in themselves are slight. There is no myosis, no paradoxical reaction, no hippus.

In mania, abnormalities are likewise found to occur frequently. There is a maximum of a weak accommodation and a minimum of a weak light reflex. There is no myosis, no mydriasis and no paradoxical reaction.

Marandon de Montyel also studied the influence of age upon the disturbances and deformities of the pupils in the insane. He made an analysis of seventy-seven cases. Between the ages of twenty and thirty, 16.1 per cent. were normal, while 83.9 per cent. were abnormal. Between thirty and forty, 21.2 per cent. were normal, while 78.8 per cent were abnormal. Between forty and fifty, 11.1 per cent. were normal, while 98.9 per cent. were abnormal. Between the ages of fifty and sixty, 11.1 per cent. were normal, while 88.9 per cent. were abnormal.

	20–30	30–40	40–50	50–60
	Per cent.	Per cent.	Per cent.	Per cent.
Disturbances	5 (41.6)	10 (3.4)	7 (38.8)	10 (55.5)
Deformities	1 (8.3)	3 (10.3)	5 (27.7)	0
Disturbances and deformities	4 (33.2)	9 (30.9)	4 (22.2)	6 ([illegible])

	20–30	30–40	40–50	50–60
	Per cent.	Per cent.	Per cent.	Per cent.
Inequality	6 (50)	12 (41.3)	6 (33.3)	5 (27.7)
Myosis	1 (8.3)	1 (3.4)	3 (16.6)	5 (27.7)
Mydriasis	4 (33.2)	4 (13.6)	2 (11.1)	2 (11.1)
Deformities	5 (41.6)	12 (41.3)	9 (49.9)	6 (33.3)
Light reflex	4 (33.2)	11 (37.9)	8 (44.4)	11 (61.1)
Accommodation	1 (8.3)	6 (20.6)	6 (33.3)	8 (44.4)
Argyll-Robertson	2 (16.6)	8 (27.2)	4 (22.2)	7 (38.8)
Reversed reaction	0	3 (10.3)	1 (5.5)	3 (16.6)
Paradoxical reaction	0	0	1 (5.5)	0
Hippus	1 (8.3)	1 (3.4)	1 (5.5)	0

The frequency of variations in the light and accommodation reflexes is shown in the following tables:

LIGHT REFLEX.	20–30	30–40	40–50	50–60
	Per cent.	Per cent.	Per cent.	Per cent.
Exaggerated	1 (25)	0	1 (12.5)	0
Diminished	3 (75)	7 (63.8)	3 (37.5)	7 (63.8)
Very diminished	0	2 (18.1)	2 (25)	2 (18.1)
Lost	0	2 (18.1)	2 (25)	2 (18.1)

ACCOMMODATION REFLEX.	20–30	30–40	40–50	50–60
	Per cent.	Per cent.	Per cent.	Per cent.
Exaggerated	0	0	0	0
Diminished	0	4 (67.6)	3 (51.4)	6 (75)
Very diminished	0	1 (16.2)	1 (16.2)	1 (12.5)
Lost	1 (100)	1 (16.2)	2 (32.4)	1 (12.5)

De Montyel comes to the following conclusions: Age has no influence upon the frequency of myosis, upon the number of disturbances present at the same time, upon the intensity of changes in the light and accommodation reflexes, upon the number, frequency, and extent and unilaterality of deformities. Age decreases the frequency and intensity of the inequality, but increases the frequency of changes in the light and accommodation reflexes and the frequency of the Argyll-Robertson pupil. It is especially before thirty and after fifty that the pupils are altered in the insane.

In the immense majority of cases he found the pupils abnormal; it may be in their movements, in their shape, or it may be in both of these qualities at the same time. He also found that though the alterations are usually slight, they are in quite a noticeable proportion pronounced. Further, they are almost always persistent and often increase with time.

Because of their detailed character, the results of Marandon de Montyel have in these pages been given special prominence, but not because the author indorses them or accepts their accuracy. Unfortunately they do not bear criticism. Especially is this true in regard to the Argyll-Robertson pupil. Here his results must be wholly rejected. As Cestan and Depuy-Dutemps point out, mere weakness of the pupil in its reaction to light or variability of this reaction from day to day are not the signs which appertain to the true Argyll-Robertson symptom. The Argyll-Robertson symptom either exists or does not exist and is not subject to daily variations. If we apply this rigid interpretation to the work of Mignot, for instance, we find that Argyll-Robertson pupil was noted by him only in one case of precocious dementia of the paranoid form, and in two observations in chronic alcoholics. This gives in reality a very small proportion, besides which the possibility of syphilitic infection in the above cases was mentioned by Mignot. Mignot very properly concludes that disturbance of the light reflex, such as is characterized by a complete and persistent abolition, offers an extreme probability of a psychosis with organic lesions.

Very different, on the other hand, are the statistics of Marandon de Montyel. In a fourth of his cases, he claims an Argyll-Robertson sign, and in a fifth, simultaneous weakness of the two reflexes, light and accommodation. These statements are surprising, for they are in complete opposition to current views. Marandon de Montyel gives the following proportions as to the existence of the Argyll-Robertson sign in the insanities: Mania, 26 per 100; lypemania, 8 per 100; dementia, 54 per 100; folie circulaire, 60 per 100; affirming thus the extreme frequency of the loss of the light reflex with preservation of accommodation to distance, exclusive of all general paralysis.

Astonished at these results, Cestan and Depuy-Dutemps in turn examined a large number of cases of insanity. The examination of the pupil was always practised in a dark chamber, with the light falling directly upon the macular region; illumination of the peripheral portions of the retina may provoke only a slight pupillary reaction even in normal persons. Among other things, Cestan and Depuy-Dutemps point out the necessity for rejecting procedures of examination which can accidentally put into play accommodation. If such precaution be not taken, the hand or the screen being suddenly withdrawn, and the eye fixing itself upon a distant point of illumination, may contract only very slightly or may indeed actually dilate. Cestan and Depuy-Dutemps noted only the exact Argyll-Robertson sign,—that is to say, the complete loss of the light reflex, with integrity of the accommodation to distance. They make mention of the fact that the investigation of this sign among the insane is sometimes impossible because of their agitation, their refusal to obey, or their dementia which interferes with the careful study of the light and accommodation reflexes.

Cestan and Depuy-Dutemps excluded all cases of paresis. They studied fifty epileptics presenting various mental manifestations, thirty old men attacked with cerebral senile lesions, twelve cases of dementia præcox, three cases of intermittent mania, two cases of *folie à double form*, five cases of melancholia, sixteen cases of paronoia, sixteen cases of secondary dementia, two cases of neurasthenia with obsessions, one case of confusional insanity, one case of chronic chorea with mental symptoms, nine feeble-minded, nine imbeciles, and six idiots. All of the cases above mentioned were examined in a dark chamber by the projection of a flame of light on the macular region. In every case was the reflex to light preserved. Thus the difference in the results of Cestan and Depuy-Dutemps and those of Marandon de Montyel is absolute; it could not be greater. On the other hand, their results approximate those of Siemerling as well as those of Mignot. It is probable that if Marandon de Montyel had tested his cases according to the method of Cestan and Depuy-Dutemps, and in addition had carefully excluded all cases capable of being interpreted as tabes or paresis, or cases presenting gross lesions of the optic nerve, the difference in results would not have been so glaring.

The results of Cestan and Depuy-Dutemps show, first, that in mental affections, as in diseases of the nervous system, the presence of the Argyll-Robertson pupil means organic disease, and secondly the finding should suggest a previous history of syphilis. How important this conclusion is, we can only realize when we remember that among the insane an antecedent personal history is often impossible to obtain.

Cestan and Depuy-Dutemps maintain that the Argyll-Robertson pupil consists in the complete absence of all contraction to light with persistence of the reaction to accommodation, no matter what may be the intensity of the light and in spite of the preservation of retinal sensibility. The sign is not susceptible of quantitative variations or gradations in intensity; it either exists or does not exist. Weakness of the light reflex should, of course, be noted, and if such weakness be associated with other organic signs of the symptom group of tabes or of paresis, it assumes some value. If such a weakness is the *only* symptom present, it cannot be interpreted as an Argyll-Robertson sign without loss of all precision. The degree of the pupillary reflexes is, of course, variable under physiological conditions, not to speak of functional disturbances, and great care should therefore be exercised in assigning a special value to a mere diminution of these reflexes. In the aged, for example, the pupillary reactions are normally feeble. They can scarcely be perceived in the case of intense mydriasis or myosis observed consequent upon drug intoxication or upon exhaustion or destruction of the cervical sympathetic. Myosis of the pupils, for instance, is frequent among the aged, and by very reason of the narrowness and limited excursion of the pupils a feeble reaction may be present. Further, a feeble reaction may be the effect of an alteration in the structure of the irides, without any lesion of the nervous system. Finally it should be remembered that a diminution of the light perception by change in the retina or by an optic atrophy is accompanied by a lessening of the light reflex.

Not only is it difficult to reconcile the pupillary findings of Marandon de Montyel with common clinical experience, but especially is this the case in his grouping of the insanities according to his findings. He groups the insanities as follows: First, a group formed by dementia and circular insanity in which abnormality of the pupils is constant in some form or other; second, a group formed by mania and melancholia, in which, without being constant, abnormality of the pupils is the most common; and third, systematized insanity in which abnormalities of the pupils are completely absent. Very curiously, he finds a constant abnormality of the pupils in circular insanity, while it is merely common in mania and melancholia. If there is one fact which has been established within recent years and which is universally accepted, it is that mania, melancholia and circular insanity form *one* symptom group, and it seems unreasonable to suppose that circular insanity, the phases of which are indistinguishable from those of mania or melancholia, should present an especial peculiarity in regard to the pupillary reactions. It is only in regard to his findings in systematized insanity that Marandon de Montyel is in agreement with other observers. The suspicion is more than justified that his reason for finding no abnormalities in the pupillary reflexes in cases of paranoia is that such cases can be examined with almost the same readiness as can normal individuals, a fact which is certainly not true of other forms of mental disease.

When we turn our attention to studies by other authors, we find that evidence as to the absence of characteristic pupillary disturbances in the psychoses is practically universal. Thus the studies of Thomsen, already referred to, and made twenty years ago can be accepted to-day as still indicative of the actual facts as to the fixed pupil. Thomsen in 1885 studied the pupillary phenomena in seventeen hundred insane. His conclusions are that by far the greater number of patients who show fixed pupil are paretic; secondly, that the symptom is of great value, because it occasionally is present at the stage of the disease when the other symptoms of paresis are still but slightly developed; third, the fixed pupil in cases other than paresis is met with (*a*) in patients of advanced age (sixty to seventy years) and who present the symptoms of senile dementia; (*b*) in chronic alcoholics in rare cases; (*c*) in other forms of mental disease which present a history of previous syphilis or head injury; (*d*) under circumstances in which it is also observed in individuals psychically normal,—*i.e.*, in tabes, multiple sclerosis, cerebral focal disease, brain syphilis, cerebrospinal meningitis, tuberculous meningitis, and oculomotor palsy; (*e*) in very rare cases without ascertainable cause.

Thomsen concludes that the value in differential diagnosis of pupillary inequality and pupillary contracture without fixation of the pupils is very slight. Loss of the light reflex was present in 47 per cent. of his cases of paresis. In the non-paretic cases only 2.2 per cent. of such pupils were found.

That no special ocular symptoms are in accord with individual forms of insanity is proven not only by actual experience but also by a brief review of writings of various authors. Beginning with the manic-depressive group of insanities, we find an absence of all mention of characteristic pupillary reactions in the works of standard authors upon insanity. Mere variations in size, dilatation or contraction of the pupil are mentioned. Among writers who have especially studied the subject, we find similar statements. Thus Cowen states that in acute mania with great excitement, one may notice, though infrequently, hippus. Occasionally also a wide pupil is seen, but at the same time a normal reaction. Later, when exhaustion is pronounced, small pupils with a sluggish reaction to light may at times be seen. Again, E. M. Thomson has described a case of acute mania with dilated pupils and sluggish reaction to light. However, in the great majority of cases of acute mania, the pupils are quite normal in size, shape and reactions.

In acute melancholia, there is usually nothing abnormal, but in cases with marked toxæmic symptoms the pupil tends to become small and reacts sluggishly to light.

Austin has seen inequality of the pupils in recurrent insanities and says that it passes from the right to the left when exaltation succeeds depression (?). Ritti has seen inequality as has also Regis. Of eleven cases examined by Mignot, only eight could be examined completely. Five times inequality was noted. One case showed a disappearance of the inequality during the quiet period. The light reflex was examined in seven cases. It was never lost and in only one case disturbed. It was normal in six. Six times convergence was obtained and was normal. In three cases deformities were noted. Inequality was present in 33 per cent., altered light reflex in 28 per cent., deformities in 5 per cent. and altered convergence reflex in none.

Among cases of senile melancholia, Minck found spasmodic myosis in melancholia with hallucinations, while mydriasis existed in the terminal stage. Preston likewise found mydriasis. In melancholia with stupor, Ball and Regis found dilated pupils with loss of light reflex. Austin says that in old people with melancholia the right pupil is often larger and more sluggish than the left. Mairet and Athanassio write that loss of the accommodation reflex and persistence of the light reflex deserve to take a place in the symptomatology of melancholia; it is quite evident, however, that abnormalities of accommodation must be referred to the inattention and psychic indifference of the patient. In twelve patients examined by Mignot, the pupillary disturbances presented special features: four showed inequality, in two the inequality was not constant but was always due to changes on the same side. With the exception of one patient, in whom the light reflex could not be investigated and in another in whom the reflex was normal, the pupils reacted poorly. The reflex to convergence was normal in seven cases. In five others the results varied at different examinations. Deformities existed in eight cases. No relation was found between the state of the pupils and the variations in the mental condition.

In paranoia, ocular phenomena are quite constantly absent. This is true not only of the pupillary reactions, but also of the fundus. Thus, J. C. Connell, who studied the eyes in paranoia, states that no constant changes were found. Conditions of depression and exaltation do not furnish corresponding appearances in the fundus. Connell agrees with the great mass of observers when he states that when affections of the eye are present, they are due to some material cause such as tabes, syphilis or albuminuria in the course of which the insanity develops. Of five cases studied by Mignot, only one had pupillary changes but this case also showed cerebral arterio-sclerosis. Of seventeen degenerates affected with systematized delusions of persecution with hallucination, there were found one case of inequality, one of myosis with sluggish reaction to light, five with a diminished light reflex, and only five with deformities. Inequality was present in 54 per cent., diminished light reflex in 27 per cent., myosis in 54 per cent., deformities in 36 per cent., disturbances of convergence in none.

In delirium, confusion and stupor characteristic changes are likewise absent. Beyond variations in the size of the pupils, nothing is noted. This is true of even delirium grave as witness the case reported by Pritchard. Occasionally, however, the pupils may be unequal as in the case of amentia reported by Pilcz, in which the right pupil was larger than the left. At times sluggishness of reaction is noted as in the case reported by A. N. V. Johnson, who describes a case of acute confusional insanity following influenza in a woman of 43. The pupils were moderately dilated and sluggish to light and accommodation.

Alcoholic insanities are exceedingly interesting but likewise show nothing characteristic. Thus, Minck found in delirium tremens, myosis and during the terminal phase, mydriasis. Ball and Regis noticed not only frequent myosis, but also inequality. In the course of the attacks of delirium with hallucinations which lead the chronic alcoholic toward dementia, Magnan has often seen inequality of pupils. The latter he has also seen in degenerates who had attacks of delirium under the influence of alcohol. Krafft-Ebing mentions also dilatation of the pupils. Mignot found that of seven patients affected with delirium and hallucinations, examined at the beginning or during the disappearance of the symptoms, only two showed no pupillary disturbances. In four cases there was found inequality; in one of them the inequality disappeared under the influence of abstention from alcohol. In two patients the light reflex was diminished; in one it subsequently again became normal. In four patients deformities were observed. Fourteen per cent. of Mignot's acute cases presented inequality; fifty-seven per cent. diminution of the light reflex, fourteen per cent. deformities, while none of them presented alterations of convergence. Among the chronic alcoholics studied by him, one only had normal pupils; six showed inequality; two suffered from loss of the light reflex; five from diminished light reflex. In one case a diminished convergence reflex was observed, in another myosis with inequality. All except one showed irregular pupils.

Raimann in 1901 published a study of alcoholic palsies of ocular muscles. He states that ocular muscles are not infrequently paralyzed in alcoholic cases characterized by Korsakow's psychosis. Statistically he found that 30 per cent. of such cases presented ocular troubles, such as inequality and impaired reaction of the pupils. A. Westphal noted paralysis of the eye muscles in a case of Korsakow's psychosis. The case was that of a man of thirty-six. The eyes could not be moved upward or downward. Visual acuity was diminished, the left pupil was larger than the right. The light reflex was sluggish on both sides, while the consensual reflex was also slight. The ophthalmoscopic examination revealed a slight pallor of the disks on the temporal sides. J. Turner has made an analysis of twelve cases of Korsakow's psychosis. In eight cases there were no eye symptoms. In four the following conditions were noted. In the first, a woman of 53, the pupils were equal and contracted, and either rigid to light and accommodation or at most reacted very slightly. In the second case, a man of 29, the pupils were equal and reacted to light with a restricted range. In the third case, a woman of 39, the right pupil was larger than normal. In a fourth case, a woman of 40, the right pupil was larger than the left; both pupils reacted to accommodation, but the left seemed rigid to light, and the right reacted very slightly to both direct and consensual light stimulation.

Regarding the pupillary reflexes of dementia præcox, definite knowledge is still wanting. According to Kraepelin, the pupils are frequently strikingly dilated, especially in the stages of excitement. This I think is undoubtedly in accord with common experience. Bumke, who has records of thirty-three cases, of which nine were hebephrenics, twenty-two catatonics and two of the paranoid form, states that in all of them the pupils were throughout larger than normal. They averaged six and one-half mm. Rarely was the pupillary diameter less than five and one-half mm. In two cases, however, nine mm. were measured. Inequality of the pupils was observed only three times and a marked difference between the right and left, over two mm., was only observed once. He observed remarkably rapid changes of the diameter of the pupils in six of his cases. Such indeed, for example, that upon one occasion five and one-half mm. were measured and several days later under the same conditions seven mm. were measured. Bumke does not venture to assign a reason for this phenomenon. A difference as to the size of the pupils does not seem to obtain among the three forms of dementia præcox.

Changes in the light reflex are doubtful in character. Kraepelin, for instance is unwilling in view of the possibilities of error in the examinations, to make definite statements as to this point. Bumke believes as far as catatonia is concerned, that if disturbances of the light reflexes are present, they are always insignificant. He has frequently received the impression that the pupillary contraction in catatonic cases comes on more suddenly and disappears more suddenly than in normal cases. He does not, however, regard the symptom as of value. He regards the reflex as active, but does not venture to say that it is regularly exaggerated.

The accommodation reaction appears to be entirely undisturbed. This is also true as to the relationship between the light and convergence reaction. On the other hand the Piltz reaction of the pupil frequently departs from the normal. It seems to be more pronounced and more readily observed than normally. For instance, in eighteen of Bumke's cases and in all of the catatonics, a contraction from two to three millimetres was readily observed. Bumke does not venture to give an explanation of this condition.

The condition which Bumke would regard as typical for dementia præcox is the absence of the psychic reflex, the reduction of the pupillary motility and the absence of the reflex dilatation to sensory stimulation. In hebephrenics, the psychic reflexes—*i.e.*, the movements of the iris in response to the play of concepts and emotions—were always absent in cases in which the disease had lasted for a relatively long time and when distinct psychic enfeeblement had become established. In two cases, Bumke noted the presence of these reactions at an early stage and later noted their disappearance. He states that he has never observed a return of this reflex in this form of dementia præcox.

In melancholia, abnormalities are very frequent but not constant. There is frequently a tendency to unilateral myosis or unilateral mydriasis, furthermore none of the patients that he observed escaped without persistent psychic defect. In three of his cases, the reflex dilatation to sensory stimulation was preserved at least when very painful stimuli were employed; twice such preservation was temporary and once it was permanent. In one of these patients, this reflex dilatation was preserved in the beginning of the disease only. In another patient, in whom it had been absent, it appeared when the psychic symptoms improved.

In the catatonic form of dementia præcox, Bumke has never been able to confirm the existence of the psychic reflexes. In six of his twenty-two cases, however, he was able to bring about a feeble dilatation of the pupils through very painful sensory stimulation. In one of the two cases of the paranoid form, both the psychic reflexes and the reflex dilatation to irritation was wanting. In the other the pupillary psychic reflexes were wanting while the sensory stimulation was still active in producing dilatation of the pupil.

Bumke also tested the action of cocaine, hematropine and eserine in dementia præcox. Definite results, however, were not obtained. Homatropine and eserine act exactly as with healthy individuals. Cocaine appeared at times to have a slight action. However, nothing definite can be said concerning this point.

It would appear, then, that in dementia præcox definite changes in the pupillary reactions are not found save, first, such as can be attributed to defective innervation,—*i.e.*, the dilatation of the pupil; and second, such changes or deficiencies of action as depend on diminished psychic activity. Definite changes in the light and accommodation reflexes are not present. Loss of the psychic reflexes occurs of course in other forms of dementia, notably in paresis. Here, however, definite changes in pupillary reactions make their appearance either early or concomitantly.

In secondary dementia, the pupils are usually quite normal, though in middle-aged cases there is not infrequently a sluggish reaction to light with a rather small pupil. In senile dementia, the only symptom noted is, as a rule, a small pupil with a distinctly sluggish reaction to light. Occasionally unequal pupils are found. In dementia depending upon gross lesions, such as tumor, softening or meningitis, the pupillary reactions may or may not be altered. If changes be present, they depend, other things equal, upon the site and the nature of the lesion and these are so various that they cannot be considered here.

SUMMARY.

In summarizing the ocular symptoms of the functional psychoses, we have to repeat the opening paragraph of this section. First, the ocular symptoms are relatively unimportant and possess but little clinical value; second, there are no pupillary changes which are peculiar to any mental disease.

In all cases in which actual and persistent pupillary changes are found to exist, we can predicate the presence of grave organic disease.

Many of the elaborate studies that have been made have failed of definite result. With the exception of the loss of the psychic reflexes noted in dementia præcox and in other forms of dementia, all of the elaborate studies as to pupillary changes in the functional psychoses may be regarded as having established nothing save the single fact that in stages of excitement and stages with marked diminution of innervation, the pupils become dilated. However, even this generalization can be regarded as having only a limited application. It is true that a dilated pupil is particularly frequent in the affective psychoses, but even here no rule can be formulated.

Because of the etiological relationship between degeneracy and insanity, we meet with—though it must be admitted *rarely*—morphological peculiarities. Though rarely present, they are always significant of arrest or malformation. We may, for instance, meet with unusual prominence of the supraorbital ridges, unusual narrowness of the space between the two eyes, or on the other hand unusual width of this space, so that the eyes seem to be too close together or too widely separated. Again, the palpebral fissures are sometimes found unusually wide or unusually narrow, or a little too straight or perhaps somewhat oblique or Mongolian in direction. Rarely gross defects, such as coloboma, may be noted. The iris may also be congenitally absent, or there may be several openings, or the pupils may be abnormally situated. Again, there may be irregularities of the pupils or perhaps an imperforate iris, or there may be anomalies of coloring, such as patches of pigment, or albinoism may be noted. The pupils may be congenitally asymmetrical and unequal. Congenital disease of the crystalline lens may be noted. While the anomalies and malformations here mentioned should be borne in mind, it is on the whole surprising that they are not met with more frequently, especially when we bear in mind the obvious relationship existing between some forms of mental disease and defective organization.

CHRONIC ALCOHOLIC INTOXICATION.

The ocular symptoms of chronic alcoholic intoxication consist chiefly in disturbances of vision. It has long been known that the chronic abuse of alcoholic stimulants may result in a more or less pronounced amblyopia. It would appear that this amblyopia is dependent upon an interstitial inflammation followed by an atrophy of the nerve-fibres. The early symptoms are those of dimness of vision, and it is worthy of note that the patient can read better in a somewhat subdued light,—that is, a light that is not very bright. Vision is, as a rule, somewhat better at night. There is present a central scotoma for colors and for white. The impairment of sight comes on gradually. Visual acuity is below normal in both eyes. Occasionally, however, the amblyopia comes on rapidly, or there may be a sudden accentuation of symptoms attendant upon renewed excesses or upon undue fatigue. According to Uhthoff, the scotomata are at first for red and green. Terrien states that the first color affected is green, and it is only much later that blue and yellow disappear. The ophthalmoscopic examination reveals in the beginning, even when vision is already much disturbed, a normal eye-ground, though in some cases there may be a hyperæmia of the papilla. When, however, the scotoma for colors and for white is extensive, atrophy of a part of the papilla may be noted. According to Uhthoff, the changes which belong to chronic alcoholic intoxication as well as those of tobacco and arsenic, consist of a partial retrobulbar neuritis. The process remains limited to the papillomacular bundle of fibres. The inflammatory manifestations in the papilla are usually slight, while the secondary

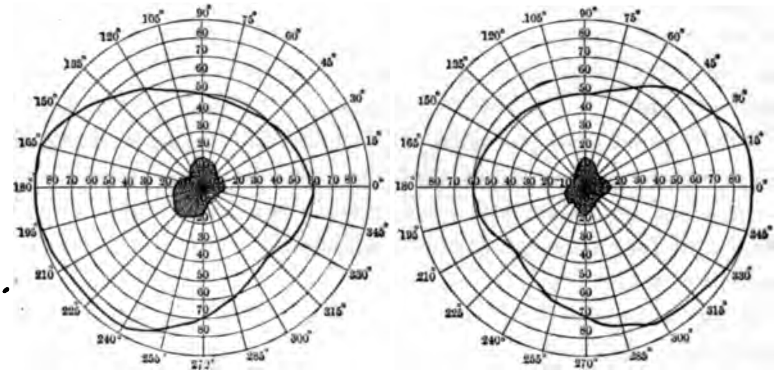

FIG. 1.—Absolute central scotomata; peripheral boundaries of the field normal in extent. (De Schweinitz.)

atrophy is usually confined to the temporal half. There is a pallor of the temporal portion of the papilla, or pallor of the lower and outer quadrant. According to Uhthoff, there is a pallor of the temporal side of the papilla in 63 per cent. of the cases. A complete optic atrophy is very rare.

According to all investigators—Erisman, Leber, Samuelsohn, Nettleship, Vossius, Magnan and Uhthoff—there is an inflammation of the interstitial connective tissue of the optic nerve analogous to that which takes place in the liver in alcoholic subjects. The fibres of the macula seem especially to suffer from the alcohol. The change consists in an overgrowth of the interstitial connective tissue, which is followed by a secondary degeneration and atrophy of nerve-fibres. Noel, however, thinks that the primary change is after all to be sought for in the nerve-fibres and that this is secondarily followed by an overgrowth of interstitial connective tissue,—that is, he believes that there is a parenchymatous neuritis, with later a secondary connective tissue proliferation and atrophy.

Uhthoff found 0.43 per cent. of alcoholic amblyopia in thirty thousand eye cases. Adler found 0.42 per cent. in seventy-one thousand eight hundred and sixty-five cases. It is remarkable that, in three hundred and four cases of alcoholic amblyopia, only four occurred in women.

The percentage of cases at various ages Adler found as follows: Between thirty and forty, 18 per cent.; between forty and fifty, 46 per cent.; between fifty and sixty, 26 per cent., and between sixty and seventy, 8 per cent.

Fleet reports a case occurring in a woman who, after imbibing a large quantity of alcohol, became blind. Eight weeks after the onset of the blindness, an examination revealed the following: The pupils were dilated *ad maximum;* there was no reaction to light; vision in the right eye consisted in the ability to count fingers at a distance of two feet, while in the left eye there was barely a perception of light; the optic nerves were dead white, while the blood-vessels were small. Fleet says that chronic subacute retrobulbar neuritis in persons addicted to alcoholic beverages is not uncommon, and we may expect a gradual or passive inflammation of the optic nerve from a gradual alcoholic poisoning. It results from a bilateral interstitial inflammation of the orbital portions of the optic nerves. The first symptoms are those of failing vision; the patient can see better in a subdued light; there is a peculiar stare; the pupils are slightly dilated; the light reaction is sluggish; the peripheral vision is better than the central, and is inability to distinguish red and green in the centre of the visual field. In acute retrobulbar neuritis, we find the pupils widely dilated; there is no light reflex, and there is no perception of light.

In acute alcoholism, visual phenomena are infrequent and with rare exceptions they are transient in duration. Interference with the visual field is almost never noted. On the other hand, paresis of external ocular muscles and disturbances of the pupillary reactions may be met with. Now and then, owing to damage to the nuclei of the nerves supplying the extrinsic muscles, a permanent paralysis may result. Blindness has, very rarely, been noted in acute alcoholism, but usually with a recovery of vision. Noel states that in acute alcoholic amblyopia there may be a rapid loss of sight in both eyes following the excess or overindulgence. Blindness may be complete from the start, but sight may later be recovered. The pupils may be much dilated and motionless.

The reflex to light may be impaired or lost during profound alcoholic intoxication but may reappear subsequently. Hans Gudden thinks that the reaction of the pupil to light is a good test for determining the state of consciousness. The light reflex may, of course, be impaired or even lost in chronic retrobulbar neuritis.

Raimann has made a study of the ocular palsies in alcoholism, and believes that the ophthalmoplegia occasionally noted is due either to a neuritis or to a polioencephalitis superior. In two cases reported by him, there was in the first a right ophthalmoplegia, both internal and external; on the left side the abducens only was paralyzed; the palsies gradually disappeared. In the second case, the pupils were small and of the Argyll-Robertson type. There were conjunctivitis, nystagmic movements, ophthalmoplegia interna, and double abducens paralysis. The autopsy revealed a hemorrhage in the aqueduct of Sylvius. Altogether, about twenty-three cases of polioencephalitis superior have been reported in which alcoholism was present. Out of forty-four cases of alcoholism studied by Raimann, there were only seven with distinct eye-muscle palsies. Although there were polyneuritis symptoms in these cases, Raimann is of the opinion that neuritis cannot be considered as explaining the palsies of the eye muscles, but believes the latter to be central in their origin.

A case with autopsy, reported by Bonhoeffer, corroborates this view. Of the extrinsic muscles the external rectus is the most frequently affected. In slight cases there is usually merely a weakness of this muscle. Nystagmic movements may be noted when the patient attempts to look toward the side of the paralyzed muscle. Next in frequency is involvement of the internal rectus. Ptosis also is not infrequently noted. These palsies usually come on suddenly.

Bonhoeffer has reported a case of alcoholism with focal cortical symptoms in which, before death, disturbances in the eye muscles occurred. Jolly has reported twenty-one cases of alcoholism, of which two presented paresis of eye muscles.

Thomsen examined six hundred and seventy alcoholics. He found no light reaction on either side in nine cases, and in one case loss of the light reaction upon one side. There was sluggish reaction on both sides in fifty-seven cases and on one side in eleven cases. In 12 per cent. of his patients there were pupillary disturbances. Siemerling found, in twenty-nine cases of alcoholism in women, only one case of loss of light reflex. Moeli, in a series of cases, found only four of permanent and three of temporary loss of the light reaction.

Raimann's experience, based upon two hundred and seven cases of alcoholism, yields twenty-five per cent. of disturbances of the innervation of the eye muscles. Among all of the eye symptoms, pupillary disturbances were the most frequent. Myosis, mydriasis, or slow and unequal reaction of the pupils, may be present. Pupillary involvement may, of course, be present at the same time that the extrinsic muscles are involved. Raimann believes that alcoholic ophthalmoplegias are almost all of central origin. The possibility of a polyneuritis of the nerves supplying the eye muscles must, however, be admitted. The prognosis of alcoholic ophthalmoplegias is uncertain.

As might be expected, amblyopia not infrequently occurs in persons who are at one and the same time the victims of chronic alcoholic and chronic tobacco poisoning, and in such cases it may be difficult and indeed impossible to separate the two conditions. It is well known that both alcohol and tobacco may independently—and this is true also of arsenic—cause an amblyopia with central scotomata and temporal discolorations of the papilla, while the peripheral vision is intact.

OCULAR SYMPTOMS OF LEAD INTOXICATION.

Blindness, as a result of lead intoxication, has long been known, though it was not until Grisole, in 1835, and shortly after Tanquerel des Planches, in 1839, described the cases studied by them that the rôle of lead in the production of blindness, together with lead colic and other general symptoms, was properly appreciated.

As in the case of alcohol, chronic lead poisoning may give rise to both amblyopia and disturbances of muscle innervation. The eye symptoms are, however, always (or nearly always) preceded by the general symptoms of lead-poisoning. The amblyopia presents typical central scotomata. At the same time, there may be concentric contraction of the fields for form and colors. Not infrequently the scotoma assumes the form of a hemianopsia.

Actual organic changes—such as optic neuritis, neuroretinitis, optic atrophy, or changes in the vessels (perivasculitis)—may supervene. Exceptionally the amblyopia is recovered from and is then probably due to an anæsthetic effect of the poison, rather than to organic changes. Ophthalmoscopic appearances, as may be surmised, vary from negative findings or mere vascularity of the nerve head to the decided organic changes just detailed. Of sixty-four cases analyzed by de Schweinitz, thirteen presented optic neuritis, four neuroretinitis and seventeen optic-nerve atrophy. In seventeen cases of the sixty-four, blindness was noted without description of the ophthalmoscopic appearances. De Schweinitz thinks that no doubt in many of these atrophy or neuritis was present, and he regards Stood's result of fifty per cent. of organic nerve change, based on a study of thirty-four cases, as not too high. Some authors—for example, Hirschberg and Knies—consider lead amaurosis identical with uræmic amaurosis. Most writers regard the influence of lead upon the optic nerves as direct. Others, like Oliver, regard the action of lead upon the retina as anæsthetic; while others still, such as Jaccoud and Weber, lay stress upon the circulatory disturbances. Elschnig has reported a case in which the circulation of the retina seems to have been at fault; there was an arterial spasm and the blindness was attributed to the ischæmia.

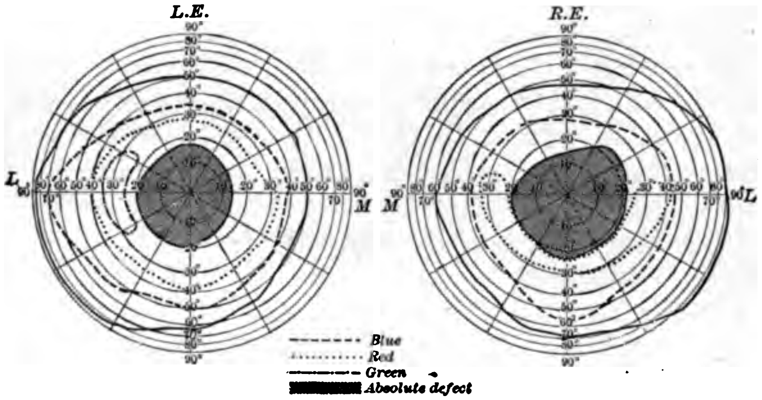

FIG. 2.—Field of vision from Uhthoff's case of lead-amblyopia with ophthalmoscopic appearances resembling intoxication-amblyopia (temporal half of papilla discolored). Normal form field and absolute central scotoma. (De Schweinitz.)

A clear conception of the visual and ophthalmoscopic findings is afforded by the following illustrative cases taken from the literature:

Westphal, in 1888, reported a case in which there were hemianopsia, cataract of one eye, and optic neuritis in the other. Hertel, in 1890, reported a case in which there was a left homonymous hemianopsia, narrowness of the retinal arteries, and sluggish reaction of the pupils. Elschnig reported, in 1898, a case of temporal hemianopsia. Bihler reported, in 1899, the case of a compositor, thirty-nine years of age, in whom in the left eye the visual field of the nasal side was lost and in the right eye the temporal visual field. The hemianopsia was partial, but there was complete hemiachromatopsia. There was slight nystagmus and the pupils were narrow on both sides. Taylor, in 1898, reported the case of a woman, twenty years of age, in whom there was failing sight with marked central scotomata in both eyes. There was white optic atrophy on the left side, while the right papilla was apparently normal; there was here an amblyopia without obvious tissue change. In two cases of lead poisoning, reported by de Schweinitz, there was partial optic-nerve atrophy together with central scotomata. In the first case, that of a house painter, aged thirty-five, vision was reduced in both eyes. The disks were nearly circular, with a sharp central excavation; there was moderate chorioidal disturbance in the circumference; there were central scotomata with contraction of the visual field of the temporal side of the right eye. In the left eye the visual field was normal; there were no muscle palsies. In the second case, also a painter, aged fifty-nine, besides marked impairment of vision, it was noted that both optic nerves were grayish, the vessels were small and there were present central scotomata for red and green. Later, there was a general grayish-white atrophy and a marked perivasculitis, with marked concentric contraction of the visual fields, especially upon the right side, and decided contraction of the red field and blindness for green.

Eye symptoms due to lead poisoning are surprisingly rare. Tanquerel des Planches found only twelve cases of amblyopia in one thousand two hundred and seventeen cases of lead poisoning. According to de Schweinitz, also, lead amblyopia, compared with the number of cases of chronic lead poisoning, is infrequent. Packard examined forty-eight workers in lead; thirty-three of them showed symptoms of the toxic influence of lead, but no case was found of amblyopia, traceable to lead. Again, lead amblyopia is rare in comparison with the whole number of ocular diseases, although it is difficult to give exact figures. De Schweinitz states that among more than twelve thousand cases of ocular disorders recorded at the Jefferson Hospital and the Philadelphia Polyclinic during five years, only three cases of optic-nerve or retinal changes attributable to lead were found and of these two were doubtful. Further, the number of amblyopias, among the total number of toxic amblyopias, is small. Uhthoff reports two hundred and four cases of retrobulbar neuritis among thirty thousand patients; of these one hundred and thirty-eight were toxic in origin, but only one was due to lead. The ocular manifestations of chronic lead poisoning may consist of transient amblyopia due to the anæsthetic effect of lead upon the optic nerve and retina, or of a permanent amblyopia due to retrobulbar neuritis. The latter may terminate in permanent atrophy, optic neuritis, or neuroretinitis specifically due to lead and which last may in turn be followed by optic-nerve atrophy. Finally, amblyopia may be due to a vasculitis of the retinal vessels.

The least known of the ocular symptoms due to lead poisoning are the disturbances of the movements of the eyes and the reactions of the pupils. De Schweinitz states that chronic lead poisoning is a well recognized cause of paralysis of the external ocular muscles. According to Schroeder, the abducens is the one most frequently involved, a condition which is, perhaps, analogous to the palsies of the extensors of the extremities.

Elschnig has described seventy-six cases of palsies of the motor nerves of the eye. Renaut, Galezowski, Dejerine-Klumpke, Mannaberg, Lilienfeld, Lagrange, and lately Aurant and Burnot, have described double palsies of the external recti. According to Elschnig, there are two varieties of ocular palsies occurring in the course of lead intoxication: First, palsy of central origin, associated with vertigo, headache, and vomiting, the cases terminating in rapid death; second, palsies of peripheral origin. The latter are more frequent, less rapid in their onset, and are less fatal. Ptosis, strabismus and diplopia may be present. The third nerve may be involved in its entirety, or the paralysis may affect only one of its branches. Isolated pupillary and accommodation disturbances do not frequently come under observation. Elschnig twice found unilateral palsy of the sympathetic, narrowness of the palpebral fissure, and sluggish light reaction. In one case he found symptoms of irritation of the sympathetic. In a case reported by Redlich there was pupillary rigidity to both light and convergence, while vision was preserved. This finding, however, must be regarded as exceedingly rare,—so rare, indeed, as to be open to doubt. Crooke has placed on record a case of lead poisoning accompanied by delirium, coma, and convulsions, followed by blindness. In this case the pupils were at first contracted and later dilated. According to de Schweinitz, the iris movements vary with the condition of the optic nerve and retina from absolute immobility to normal reaction.

CHAPTER XIII.

AMAUROTIC FAMILY IDIOCY AND CEREBRAL PALSY OF CHILDHOOD.

BY B. SACHS, M.D.

AMAUROTIC FAMILY IDIOCY.

THE above title was given by me to a rare disease affecting several members of the same family, and characterized by a distinct lack of mental development, by a progressive weakness of all the muscles of the body, and by a defect in vision (associated with changes in the macula lutea and optic nerve atrophy) terminating in complete blindness. The disease is generally fatal, the children dying as a rule in a condition of complete marasmus before the end of the second year of life.

History.—In 1881 Waren Tay described a case presenting "symmetrical changes in the region of the yellow spot in each eye of an infant. The child was twelve months old. It was deficient in holding up its head or moving its limbs. There was weakness but no absolute paralysis of any part. Its cerebral development was slow and poor. At the first examination, March 7, 1881, the optic disks were apparently healthy, but in the region of the yellow spot of each eye there was a conspicuous, tolerably diffuse, large white spot more or less circular in outline, and showing at its centre a brownish-red, fairly circular spot contrasting strongly with the white patch surrounding it. This central spot did not look at all like a hemorrhage, nor as if due to a pigment, but seemed a gap in the white patch, through which one saw healthy structures." The author likened these appearances to those one is familiar with in cases of embolism of the central artery of the retina. He believed the changes in the retina to be "possibly congenital." Five months later another examination was made, showing that the disks had become atrophied, but that the changes in the macula lutea were the same as before. In the same family, according to Waren Tay's later reports, three similar cases had occurred, each one of the children presenting ocular symptoms and exhibiting physical conditions that were similar in all respects, and all three dying before the age of two years. This peculiar ophthalmoscopic finding was noted by Magnus, Goldzieher, Wadsworth of Boston, Hirschberg of Berlin, and H. Knapp.

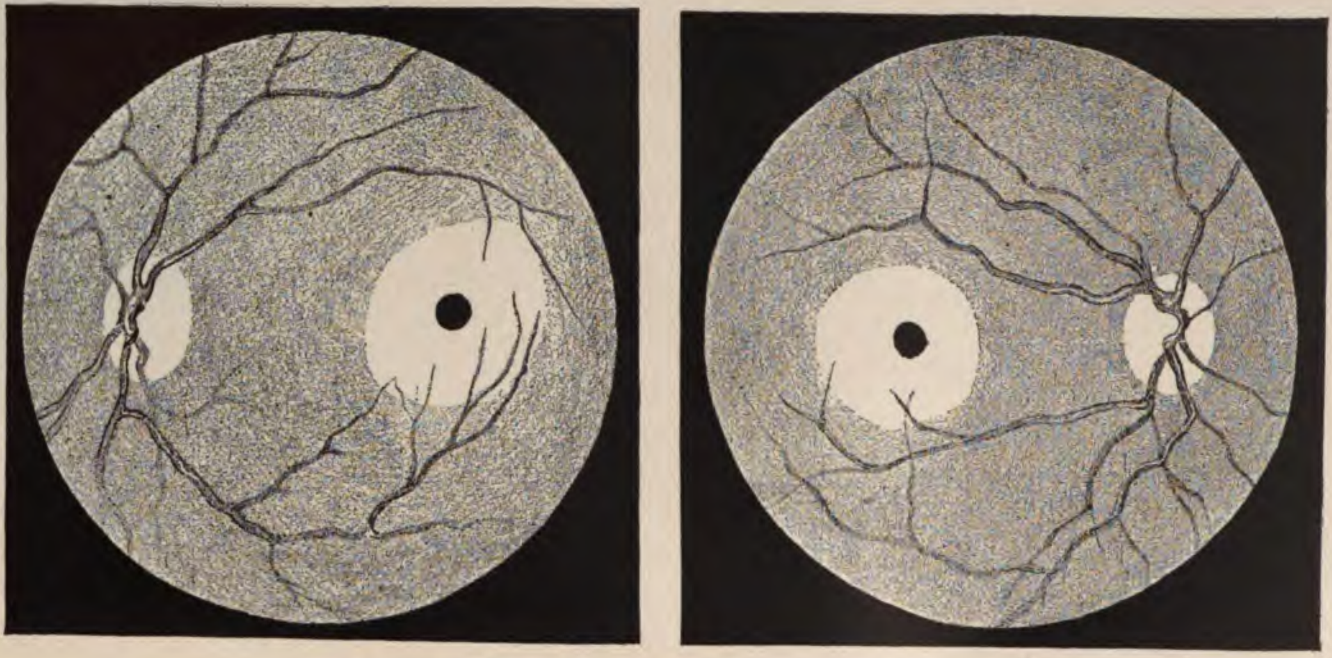

Fig. 1.—Amaurotic family idiocy. Showing characteristic changes in the retina.

In 1887, without any knowledge of the cases described by the oculists, I published the history and the post-mortem record of a patient suffering from what appeared to be a peculiar form of idiocy associated with blindness. The family character of the affection was not evident until a sister of my first patient became similarly affected. In still another family I saw another instance of this affection, and received the history of three other children who had been afflicted with and had died of this disease.

Kingdon, of Nottingham, called attention to the fact that the rare condition reported by the oculists was part of the disease which I had described. In 1894 Carter collected all cases of this disease known up to that time, and in 1896 I was able to give a list of nineteen cases of which eight had come to my own notice. Since the publication of this last paper other cases of this sort have been described and published by American and some European writers. In Europe cases have been published by Kingdon and Russell,[1] Higier, Falkenheim, Shaffer, Frey, and others. In 1901 Falkenheim analyzed a series of sixty-four cases and since that time others have been reported both here and abroad.

Symptomatology.—As a rule, the children affected with this disease are born at full term and apparently in perfect health. They do well until the first three to six months of life, when they become listless and apathetic, move their limbs very little, and show the first signs of visual disturbance which ultimately leads to blindness. The child is not able, as the months go on, to hold up its head or to sit up. Its muscles are either flaccid or spastic; the reflexes are normal, a trifle subnormal, or exaggerated. In some cases there is an unusual sensitiveness to touch and to sound (hyperacusis), the child being startled by the slightest noise occurring in the room. Convulsions are present in some cases but are not an integral symptom of the disease. All the functions of the body are in a low state of activity. The children are subject to frequent bronchial attacks and soon show gastro-intestinal disturbances. An examination of the fundus reveals the peculiar condition so well described by Tay. (See Fig. 1). There is a gradual increase of all the symptoms, the mental defect becomes more and more noticeable, the palsy more extreme, complete blindness is established and the child gradually lapses into a condition of marasmus in which it dies as a rule before the end of the second year. The chief symptoms may be summed up as follows: (1) Mental impairment observed during the first months of life and leading to absolute idiocy; (2) paresis or paralysis of the greater part of the body, and this paralysis may be either flaccid or spastic; (3) the reflexes may be normal, deficient or increased; (4) a diminution of vision terminating in absolute blindness (the cherry-red spot in the region of the macula lutea and later optic nerve atrophy); (5) marasmus and a fatal termination, as a rule, before the age of two years; (6) the occurrence of the affection in several members of the same family.

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[1] Russell has used the designation "Infantile Cerebral Degeneration;" the term is unsatisfactory and wholly inadequate.

In some, but not in all of the cases, nystagmus, strabismus, and hyperacusis are added to the above cardinal symptoms. A loss of the sense of hearing was noted in two cases. Falkenheim refers to impulsive laughter, but I have not been able to persuade myself that this had occurred in any except in the one case described by him. Some slight variations will naturally occur in the degree and character of paralysis. The changes in the macula lutea are so striking that they constitute a very important symptom of the disease. But it is to be noted that the disease can be diagnosticated even in the absence of this one symptom, and that in some of the cases the general cerebral symptoms are developed some months prior to the retinal changes. Koller has reported a case in which at the first examination the changes in the macula lutea were not in evidence although they appeared later on, and in Higier's case the optic nerve atrophy was much more pronounced than the changes in the macula lutea.

Etiology.—The causes underlying this disease are still obscure. In some of the families there has been blood relationship between the parents, in many others no such relationship existed. Injury to the mother during pregnancy has been noted in several of the cases and the tendency to mental derangement in the families of one or both parents is also suggestive of another possible factor. The family predisposition is evident from the fact that twenty-eight cases have occurred to my knowledge in fifteen families. Carter was the first to call attention to the fact that all of the cases reported have occurred among Hebrews, and even at this day I can say that I know of no indubitable case occurring in others. The racial feature of the disease is all the more astounding, because other diseases to which it must necessarily be more or less closely allied have been observed and recorded among all races and all nationalities.

The absence of syphilis has been distinctly noted in most of the histories. This is a matter of some importance for there is always a tendency to attribute a mysterious family affection to this hereditary factor, and furthermore, the disease bears some resemblance to specific disorders which are characterized by various forms of dementia and by ocular disturbances (Batten). Starr was mistaken in quoting the present writer as favoring the syphilitic etiology in this disease. In passing we may take note of the fact that there is an hereditary optic nerve atrophy occurring later in life which is due in many instances to hereditary syphilis, and with this optic nerve affection the present disease might possibly be confounded.

The disease runs its course, as was intimated above, in a little less than two years. I have encountered but a single prominent exception to this rule, and in this instance the child had attained the age of five and a half years when I examined it, and it is, so far as I know, still living. The characteristic symptoms of the malady were present, and the same symptoms had been presented by two or three other members of this family. Spiller threw out the hint that the symptoms and the anatomical changes may vary somewhat if some of these children should live beyond the usual term of years. Many of these children are extremely well nourished at birth and for the first few months of life. By degrees they lapse into a state of marasmus to which they slowly succumb. I wish to insist on marasmus as a very important symptom of the condition, and the emaciation observed in such cases is so extreme that it unfortunately makes of the child a most disagreeable object. One writer has referred to sudden death as characteristic of the disease, but the very opposite of this has been observed in my experience.

Pathological Anatomy—In my first case of palsy, in 1887, the outer surface of the brain exhibited abnormalities which we were accustomed to associate with brains of inferior development. There was a confluence of the central and Sylvian fissures and a complete exposure of the island of Reil. There was unusual hardening, the knife grating on removing a small section of the cortex. On microscopical examination the most important changes were found in the cortex, and sections were taken from the frontal lobes, motor areas, from the base of the 3d convolution, from the first temporal, and from a part of the apex. The same changes were found practically throughout the cortex. It was possible to make out the various layers of cells, but examination and a most careful search on removal of the sections showed that there were scarcely half a dozen pyramidal cells which presented anything like their normal appearance. The contour of the cells was either rounded or elongated, and the cell protoplasm exhibited every possible change such as we note in degenerated cells. In some cells the nucleus and necleolus were entirely wanting or were relegated to the margin of the cell. In later examinations further changes had been noted. Hirsch was the first to point out that these same cellular changes occurred not only in the gray matter of the cortex, but in the gray matter of the entire central nervous system, in the cortex of the brain, in the basal ganglia, in the gray matter of the spinal cord, and even in the spinal ganglion. I was able to corroborate this finding by a later examination in another case of my own, and in 1903 I felt warranted in declaring amaurotic family idiocy to be a disease chiefly of the cortex and of the gray matter of the entire central nervous system. These findings have received further and most satisfactory corroboration in a careful report recently published by Spiller. Kingdon, Schaffer, and others, including Hirsch and myself, have found degeneration in the pyramidal tracts, yet this degeneration does not seem to be as marked as we would expect it to be from the very considerable involvement of the gray matter.

Holden, Mohr, Treacher Collins, Schumway, and Buchanan have examined the eyes post-mortem. There is a degeneration of the ganglion cells of the retina, and of the nerve fibres of the optic nerves and tracts. There is some doubt as to the existence of an œdema in the region of the macula.

The question has arisen whether the disease is invariably congenital, or whether it is an acquired disease. The latter is claimed to be the case by Hirsch, who would place it in the category of toxic affections; but, as I have stated in several communications, it is hard to accept such a view for any family disease, and the fact that it cannot be attributed to the influence of the mother's milk is proved by the one circumstance that several of my patients were raised by strange wet nurses. There is no doubt in my own mind that the disease is developed as the result of a congenital defect. The nervous system of such a child is able to perform tolerably normal functions until the age of four or six months, then it is no longer able to meet the strain put upon it, and an active process of degeneration sets in. This degeneration is, to my thinking, a natural result of an arrest of development. Gowers has claimed very much the same for some of the scleroses that have developed very much later in life, and the fact that the first symptoms of amaurotic family idiocy become evident about six months after birth, does not militate against the theory of a congenital defective development.

Amaurotic family idiocy should not be regarded as an isolated though interesting disease, but all the symptoms and anatomical findings suggest a relationship to other hereditary family affections.

Treatment.—Unfortunately, little can be said in favor of any therapeutic measures. In several cases under my observation I have hoped against hope that, by most careful nursing and feeding, some of the children thus afflicted might escape, but up to the present time all such efforts have been entirely fruitless. Kingdon has suggested the use of thymus extract, but he is not yet able to state that any advantage has been gained thereby. It would be natural to infer that the wisest thing to do would be to avoid bringing such children into the world; but, as several healthy children have been born to parents who have had one or two children afflicted with amaurotic family idiocy, advice bearing upon this point cannot readily be given. In conclusion I consider it necessary to remind the oculist that these patients can no longer be regarded as afflicted with a rare ocular condition, but that the ocular condition, interesting as it is, is merely one symptom of an easily recognizable family affection.

INFANTILE CEREBRAL PALSIES (SPASTIC HEMIPLEGIA, DIPLEGIA, AND PARAPLEGIA).

Although varying widely in their mode of origin, the cerebral palsies of children deserve to be treated as a distinct clinical group of diseases. A study of the subject many years ago led me to divide them into three classes in accordance with the mode of onset,—*viz.,* those that were prenatal in origin, those that were due to difficulties during parturition, and those which were distinctly acquired some years after birth.

Whatever the time or manner of origin may have been, most of these cerebral palsies are observed in the earlier years of life,—from the time of birth up to the age of ten years, even later; but by far the greater proportion of them occur during the first three years of life. The paralysis is of the distinctly spastic order and according to the distribution of the palsy we may distinguish between a hemiplegia and a diplegia (double hemiplegia), or paraplegia. Monoplegias are relatively rare, and some of those that appear to be monoplegic in character are practically the incomplete diplegias or hemiplegias. The rigidity of the muscles, the contractures resulting from the same causes, and increase of all the deep reflexes in the paralyzed members are the constant accompaniment of these palsies. Coma and convulsions may occur in the initial stage of the acute forms, and the convulsions may be often repeated during the later stages of the disease. The cerebral palsies of children are more frequently associated with coma and convulsions than are those of the adult; the former are generally due to cortical processes, the latter to intra-cerebral conditions. To say that the convulsions may frequently be repeated is equivalent to saying that an epilepsy has been established which may continue throughout life, and the same changes which have caused both the paralysis and the epilepsy may also be responsible for the defective mental development, which may range from weakmindedness to marked imbecility and complete idiocy. It is especially to be noted that disturbances of motion, associated movements, ataxia, athetoid, choreiform, and even cataleptic movements, occur often enough in connection with these diseases.

Aphasia, a common accompaniment of the palsy of the adult, is a much rarer complication in infantile palsies. It goes without saying that all these conditions are characterized by certain negative symptoms which are of greatest importance in attempting a differential diagnosis between the cerebral spastic and the spinal flaccid palsies. Such symptoms include the entire absence of changes in the electrical reactions and the absence of any marked atrophy in association with the palsy, although a slight atrophy from disuse is common enough.

Etiology.—In discussing the origin of cerebral palsies, we must bear in mind the three groups alluded to above. As for the prenatal cases, they may be due to hereditary taints, which is equivalent to saying that such cases occur in families with a decided history of various neuroses, with a history of hereditary epilepsy, or of hereditary insanity. The next most important etiological factor is unquestionably the occurrence of some traumatism to the mother during the period of pregnancy. The manner in which such traumatism may injure the brain of the child was clearly shown by Cotard, who found in the brain of a still-born child an old lesion in the right hemisphere in the vicinity of the lateral ventricle. Illness of the mother during pregnancy, exhausting fevers, particularly such as accompany pneumonia and typhoid fever, uræmic convulsions, and severe fright, are other causes which have been made out in cases of prenatal palsy.

[illegible] influence of hereditary [illegible] been described [illegible] factor in some of [illegible]

[illegible] While the brain of [illegible] of pressure, it is [illegible] at birth, as [illegible] considered a most [illegible] labor is a [illegible] instrumental delivery [illegible] with greatest frequency [illegible]

[illegible] They may [illegible] x, and even [illegible] is very [illegible] whether the palsy is due to the effect of the toxic agent or to the mechanical injury during a spasm of coughing. Fright, which Freud has dwelt upon, as the equivalent of a psychic trauma is an occasional cause of acute cerebral paralysis, but actual injury to the skull is a much more powerful factor. These palsies occur also after a simple or cerebrospinal meningitis, after an exhausting gastroenteritis, and after other fevers. Of ninety-one cases of acquired cerebral palsy which I studied many years ago, the exact cause could not be ascertained in twenty-seven.

There was a tendency some years ago, particularly among French writers, to claim that all cases of acquired cerebral palsy were due to acute infectious diseases. This view was also insisted upon by Strümpell, who established the analogy between the acute cerebral and the acute spinal paralyses of children and concluded that the former are due to a polioencephalitis just as the latter are due to a poliomyelitis. While the dependance of acute or acquired cases of polioencephalitis has been very much over-stated, there is evidence enough to compel us to concede that some of these acquired cases may be due to an encephalitis. A discussion has arisen between other authors and myself regarding the etiological importance of convulsions. Freud and Rie are of the opinion that convulsions denote the onset of the cerebral process, but that they never hold a causal relation to the palsy which results from the cerebral lesion. While one may be willing to grant that in a very large number of cases this statement of the Austrian authors holds good, Osler and myself have advanced the view that in many others the palsy seems to be more or less the immediate result of the convulsive seizure. Anyone who has observed the marked disturbances of circulation at the acme of a convulsion, can readily conceive how easily a blood-vessel could burst during this period as a result of excessive stasis; and that such venous hemorrhages occur in epileptic convulsions I have had reason to know from the examination of the brain of a girl of seventeen years, who died three days after a severe epileptic attack. In this case there was a large subpial extravasation, which covered quite completely the entire left hemisphere and part of the right. If such an occurrence could take place in the brain of a girl of seventeen, there is no reason why similar accidents of lesser extent should not happen frequently in the case of children. There is also further clinical evidence on this point, and those who are specially interested in this subject are referred to a discussion of it in my book on "Nervous Diseases of Children," page 531.

Symptomatology.—The distribution of the paralysis may vary with the condition of the paralyzed limb and is the same in all cases. The hemiplegic form resembles in every respect the hemiplegia of the adult, and further analogy is observed in this that the leg recovers very much more quickly than the arm, just as in adult hemiplegia. Under diplegia, we may classify all those cases in which both halves of the body have been involved, and it is better to attach the greatest importance to this bilateral character of the palsy even though the palsy be incomplete, for the bilateral affection points to a double brain lesion and that is, after all, the salient point for consideration. In diplegias, the legs may be much more affected than the arms, there may be cross-legged position, and cross-legged progression, and in some instances the arms may appear to have escaped to such an extent that what is an incomplete diplegia may appear to be a paraplegia, but a paraplegia associated with athetoid or other disturbances of motion in the upper extremities could be designated as an incomplete diplegia; but whether the form of paralysis be a diplegia or a paraplegia, such paralysis must necessarily be due to a double cerebral lesion and these are almost invariably prenatal in origin or the result of traumatism during labor, and yet it is well to state that both diplegias and paraplegias also occur every now and then in the acute cerebral cases, contrary to the opinion of Strümpell, who holds that infantile *hemiplegia* is synonymous with the acute cerebral paralysis of children.

Aphasia may be associated with these infantile cerebral palsies. It is invariably motor, never sensory, in character. It need not be added that aphasia will be developed only in children who have acquired articulate speech before the onset of the paralysis. This excludes from the list all diplegias and paraplegias which have come on before or during birth. It must be remembered that a complete absence of speech development may be present in children who exhibit cerebral and mental defects and I have seen some children in whom the mental defect seemed to be due more largely to defective development of the speech centres than of any other part of the brain.

In addition to the form of paralysis, it is well to pay special attention to the rigidities and contractures. These abnormal conditions of the muscles are so prominent that doubt has arisen whether some of the diplegias or paraplegias would not be more properly spoken of as cases of congenital spastic rigidity, for the rigidity is most marked and the paralysis often very slight. In the case of Cotard quoted above in which the palsy was due to an intrauterine lesion, the child was born with contractured extremities. The contractures may vary in degree and in the number of joints affected. The flexors and pronators of the arms, flexors of the legs and of the feet are most frequently involved.

In the case of diplegia and paraplegia there is, in addition, contracture of the adductors of the thighs, which is responsible for the peculiar cross-legged progression and cross-legged position. Pes equinus or pes equino-varus is the most common deformity of the foot. In a few cases an equino-valgus is present. If the upper extremity is contractured the arm is held in close juxtaposition to the trunk, is flexed at the elbow, and the hand is generally in a position of extreme flexion, the fingers often being firmly pressed into the palm of the hand.

The post-paralytic disturbances of motion constitute a very characteristic group of symptoms in cases of infantile cerebral paralysis. While they are seen occasionally in adult apoplexy, they are far more common in the paralytic disturbances of youth. The choreiform movements are of importance because cases of that kind are often mistaken for cases of ordinary chorea, but of course the presence of paralysis or of contractions, and the exaggeration of the reflexes in the choreic extremity, will be sufficient to indicate the nature of the choreic movements. Athetoid movements are of special interest, inasmuch as they are observed invariably in connection with cerebral palsies. Associated movements are often developed to a marked degree, the paralyzed hand imitating all movements of the normal hand.

Epilepsy is, perhaps, the most serious of all conditions associated with infantile cerebral paralysis. The special point of importance is that the palsy may disappear or may be so slight as to give little inconvenience to the child, whereas the epilepsy remains. Special reason for insisting upon this point is that in such cases the epilepsy as well as the paralysis is of organic origin and we cannot therefore expect to cure the disease by the use of the ordinary measures.

Idiocy is also developed very often both in association with epilepsy and with cerebral palsies. The severer forms of mental retardation are present in cases of diplegia and in cases of paraplegia rather than hemiplegia, and this association can be explained easily enough if we remember that the diplegias and paraplegias point to a double lesion, whereas the hemiplegia results from a lesion in only one-half of the brain.

As for the eye symptoms observed in these conditions, the commonest are strabismus and occasionally nystagmus, and as Freud has pointed out, hemianopsia may be associated with the brain palsies of children. The latter condition is surely a very uncommon one and would depend largely upon the accidental involvement of the occipital region in a case of cerebral defect or in a case in which considerable blood had been exuded over the occipital region of the brain during parturition. The pupillary reflexes are normal in such a vast majority of these children that they are of no symptomatic value. Freud has reported some cases of optic atrophy associated with diplegias and he thinks that these conditions, which are rare enough, may bear a very close relation to the changes found in cases of amaurotic family idiocy. I have never seen changes in the macula lutea in any case of distinct cerebral spastic paralysis.

Morbid Anatomy.—The entire subject of morbid anatomy of infantile cerebral palsies will be sufficiently stated in this connection by the reproduction of a table which was first embodied in the chapter on these diseases in the author's text-book, and which has been very fully quoted by later authors. I have nothing to add to it, and believe that it fully represents our present knowledge of the subject.

CLASSIFICATION OF INFANTILE CEREBRAL PALSIES.

GROUPS.	MORBID LESIONS.
I. Paralyses of intra-uterine onset..	Large cerebral defects (porencephaly). Defective development of pyramidal tracts. Agenesis corticalis (highest nerve elements involved).
II. Birth palsies	Meningeal hemorrhage, rarely intracerebral hemorrhage. Later conditions: meningo-encephalitis chronica, sclerosis, and cysts; partial atrophies.
III. Acute (acquired) palsies	Hemorrhage (meningeal and rarely intracerebral); thrombosis from syphilitic endarteritis and in marantic conditions: atrophy, cysts, and sclerosis (diffuse and lobar). Meningitis chronica. Hydrocephalus (seldom the sole cause). Primary encephalitis; polioencephalitis acuta (Strümpell).

Differential Diagnosis.—First of all it is important to distinguish between the cerebral and the spinal paralyses of children, but anyone who is able to recognize the difference between brain and spinal lesions will be able to make this differential diagnosis. In the one case we have spastic paralysis, increased reflexes, normal electrical reactions, no atrophy, and generally a hemiplegic or diplegic form of paralysis. In the spinal group the paralysis is flaccid, the knee-jerks are absent, there is very marked atrophy and the paralysis may affect several members of the body, or several large groups of muscles, but is never hemiplegic. Moreover, the electrical reactions in the spinal cases are always involved and altered. Difficulties may, however, arise in some mild cases of cerebral paralysis in which the spastic contractions are but slightly developed and the reflexes not very much increased. The presence of athetoid or choreic movements in one-half of the body points to cerebral paralysis, but these are never associated with spinal palsies.

The oculist should bear in mind that the nystagmus and strabismus may be symptoms of multiple sclerosis, but the differential diagnosis should be based upon scanning speech and upon the absence of associated movements, and the post-paralytic disturbances of motion which are so common in the infantile spastic palsies. Inasmuch as there is a hereditary form of multiple sclerosis this differential diagnosis may have to be made with the greatest possible care. A very distinct hemiplegia or a diplegia would militate against the diagnosis of multiple sclerosis.

Treatment.—The treatment of infantile cerebral palsies calls for deliberate non-interference at the start. The treatment of the later stages will be practically the same whether the case be congenital or acquired. If a young infant exhibits a tendency to drowsiness or to convulsions, its brain has in all probability sustained a serious injury during labor. Strict attention should be paid to the nutrition of the child, and milk properly prepared according to the age of the child, or else breast milk, is the only food to be administered. If there is a very marked tendency to convulsions, small doses of bromide or minimum doses of morphia or chloral should be given. If convulsions continue in spite of these measures, careful inhalation of chloroform may be practised. All other measures, as counter-irritation, mustard baths and the like, are useless.

In a case of an acute cerebral palsy in a child, the fewer drugs that are administered the better. The physician will do well to limit his therapeutic efforts to the administration of calomel in doses sufficient to procure a free purging of the bowels, and small doses of bromides to secure rest for the disturbed brain. At a later period bromides may be combined with small doses of iodide, but whenever nutrition is disturbed, it is best to abandon all drug treatment. After the symptoms of the initial period have passed, the physician is called upon to prescribe some form of treatment for the paralysis and the other symptoms of the disease. After the paralytic condition of the active period of the disease has passed, the condition is to be treated by massage and electricity applied to the paralyzed parts, but let the brain severely alone. While electricity applied to the extremities can do no harm, the effect on the injured brain is altogether too uncertain. When epilepsy is developed the condition must be treated as all other organic epilepsies should be and in some of these cases, according to the experience of Horsley, Wyeth, Gerster, and myself, there is sufficient reason to attempt surgical interference, particularly if all the symptoms point to a well localized lesion, and above all, if there is a probability of the existence of an old hemorrhagic cyst, a condition which is found in not a few of the congenital and acute cases. In children in whom the rigidity is the most marked symptom, therapeutic measures are in order and tenotomies, as well as transplantation of tendons, yield good results in the hands of competent men.

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CHAPTER XIV.

ABNORMALITIES IN THE DEVELOPMENT OF THE BRAIN AND SKULL; FACIAL HEMIATROPHY; FACIAL HEMIHYPERTROPHY; ACROMEGALY.

By CHARLES W. BURR, M.D.

I SHALL not consider anencephaly or any of the conditions in which the abnormality of development or the intrauterine disease is so great as necessarily to cause fetal death. Such conditions are of great interest in the study of the causation of monsters, but are of no clinical importance. The normal development of the brain and that of the skull are closely associated with each other and pathological development in either may affect the other, but for convenience sake they are studied separately. Strictly speaking, the term "defects of development" should be used to mean only defects due to an improper growth of the brain on account of inherent deficiency in the germ or sperm cell and should not include abnormalities caused by acquired disease occurring in the embryo; but our ignorance of embryonal pathology is so great that such a distinction can rarely be made.

Apart from the abnormal development which causes gross defects there is another kind of lack of development, really congenital in origin though the symptoms of disease do not appear till some time, it may be years, after birth. I mean that condition in which the patient may at birth seem normal, may indeed pass normally through infancy, and then, or even later, show symptoms of disturbance of the functions of the brain or cord, not produced by any of the known causes of disease, but by inherent weakness and inability to withstand the physiological stress of life. This weakness may exist not only in the nervous system but in any part of the body. It has been recently described, or rather offered as an explanation (for, of course, it is a theory and not an established fact), by Sir William Gowers under the title "**Abiotrophy.**" He states, what has been long known, that life is a double thing,—somatic, that of the body as a whole, and local, that of each element by itself. Death of some of the elements may occur without somatic life being affected. This process we call degeneration, but it is really a slow decay, a local death due to an essential failure of vitality. This process may affect not only some one system of nerve-fibres in the brain or cord, as in Friedreich's ataxia or congenital cerebellar atrophy, but also the muscles, as in the various forms of idiopathic muscular atrophy. Gowers believes that there is a certain type of degeneration of the optic nerve, occurring in families, which is really abiotic, really due to an inherent weakness of the nerve-fibres and in which tobacco or some other poison acts only as an exciting, not a true, cause. This theory does not claim that all degeneration in the pathologist's sense of the word is abiotic.

Porencephaly.—The word porencephaly was first used by Herschl and the first elaborate monograph was written in 1882 by Kundrat. By them it was used to mean a hole going into the brain, not the entire absence of brain structure and not minute cavities resulting from small vascular lesions. Porencephaly is classified as congenital, coming on during intrauterine life, or acquired, occurring at or very soon after birth. Clinically the differentiation is always difficult, often impossible. Practically it does not matter to which type a case belongs. The congenital cases like the acquired are probably always due to disease and not the result of a true developmental defect.

The cavity may occur in any part of the brain but is most frequent in the anterior or middle. Usually there is only one cavity but there may be two situated in the same position in either hemisphere. For example, the parietal region may be affected on both sides or both frontal lobes. When there is only one cavity it is more apt to be in the right hemisphere. Brill states that no case has been reported involving the median surface of the hemisphere, but Brush reports a case in which the cuneus was much affected.

Kundrat holds that the immediate cause is anæmic necrosis. Schultze would sharply separate the cases of uterine origin from those occurring after birth by the following rule: In the former, he states, the convolutions are so arranged as to radiate toward the margin of the cavity or towards its centre, while in the latter the hole is irregular in outline and the convolutions have no regular arrangement. The most frequent direct cause is probably vascular or meningeal inflammation. Traumatic encephalitis may occur during intrauterine life and cause the formation of a cavity. Schultze, for example, holds that spasmodic contraction of the uterus arising from causes within itself may injure the fetal brain. Alcoholism in the mother and syphilis in either parent have been cited as causes of obliterating arteritis in the fœtus. B. Sachs speaks of the possibility of some purulent disease in the mother during pregnancy as being sufficient to cause meningitis or encephalitis which might, occurring in a growing fœtus, produce porencephaly. In acquired porencephaly the primary lesion seems almost always to be a meningeal hemorrhage occurring at birth and resulting from pressure on the skull during labor. Whether meningeal hemorrhage occurs most frequently from the use or non-use of the obstetric forceps is much discussed. Probably more injury has been done by the non-use of the forceps than by their unskilful application. Whatever may cause the hemorrhage, if it be large enough, there is compression of the brain substance, atrophy of the cortex and underlying white matter and a consequent cavity. The pia and cortex are apt to become inflamed, agglutinate, form a dense membrane lining the cavity which is filled with fluid, thus forming a cyst covered by the outer membranes. The time required for the absorption of the brain tissue and the formation of the cavity is unknown. Acquired porencephaly is most apt to occur in the parietal region and is usually unilateral, but may be bilateral.

There are no pathognomonic symptoms. It is not difficult to determine the existence of organic brain disease, but it is impossible to say with certainty whether the symptoms are due to porencephaly or to cerebral atrophy without cavity formation. Almost always, but not invariably, there is marked mental defect. Usually there is idiocy or at least imbecility. Rarely in unilateral cases without great destruction of the cerebral tissue the mind may be unimpaired. The most frequent physical symptoms are spastic hemiplegia or diplegia. Epileptiform convulsions are very frequent. If the cuneus is involved hemianopsia must result. Often there is a spastic disorder of speech and sometimes a true aphasia or rather, from the destruction of the potential speech centres, an inability to learn to speak. Probably the majority of the patients, certainly a very large number, die in early life; a few survive to maturity, and even old age.

Microcephalus.—The term is used to mean only those cases of a small brain associated with a small skull and due to defective development of the entire organ. It must not be forgotten that a skull of normal size may contain a brain of very small volume, the surplus space being filled by fluid. Any skull under seventeen inches in circumference is, according to Dr. Ireland, to be classed as microcephalic. The head is, as a rule, cone-shaped or oxycephalic. The forehead slants backward in marked manner. The palate is generally flat and the base of the skull asymmetrical. The face is never proportionally as small as the cranium. The causes of the condition are very obscure. There is no doubt that a few cases are produced mechanically by a premature ossification of the sutures preventing the growth of the brain. This was Virchow's theory. Klebs's theory that hour-glass contraction of the uterus on the fetal head is a cause is no longer regarded with favor. It is probable that some cases are caused by intrauterine cerebral inflammation. B. Sachs describes cases occurring after birth and following convulsions in which it was probable, he thinks, that the convulsions resulted in extensive meningeal hemorrhage or pachymeningitis hemorrhagica, in consequence of which there was atrophy of the brain and associated with it a lessening in the growth of the skull. In these cases the children were normal at birth and with skulls of average circumference. The relation, under normal conditions, between the growth and development of the brain and skull is very obscure. Since, however, there may be a small and universally diseased brain associated with a skull of normal size, the space being filled with fluid, it is not probable that the brain exerts any trophic influence on skull growth.

Though the entire brain is affected in microcephaly the cerebral hemispheres are most deficient. The cerebellum and the basal ganglia are less abnormal. The spinal cord, as a rule, is also shorter and smaller than normal. The columns of Goll, the anterior horns, and pyramidal tracts are especially affected. Sometimes even with spastic diplegia the cord is only small and shows no degeneration. Small as the brain may be, and Ireland reports one the weight of which was seven ounces, it is distinctly human in form, and could never be mistaken for that of one of the lower animals. The primary fissures are always present, the secondary may be absent or may be replaced by others much more numerous, making a large number of small convolutions (microgyria). In old times the normal brain was compared in appearance to a bunch of worms; the resemblance is much greater in some cases of microcephaly. In the cases in which there are fewer fissures than normal the surface of the brain is, of course, smooth. It is of historic interest that at the time when Darwinism was a new thing, Charles Vogt wrote a book to prove that microcephaly was atavistic,—was a reappearance of the simian type. This opinion has not stood the test of time.

The brain is deficient not only macroscopically but also microscopically. There are fewer nerve-cells than normal in the cortex and in the anterior horns of the spinal cord, and many of the cells present in the cortex either have no processes or greatly stunted ones. In some cases there is an overgrowth of connective tissue.

The condition is not common; indeed it ranks among the rarer forms of idiocy. It can, of course, be diagnosed at sight, but except in the cases of extreme smallness of the head, it is not always possible to predicate the extent of idiocy from the appearance of the head. In other words, barring the extremes, the idiot with the smaller head may have more intelligence than he who has the larger. The explanation of this is, that in the former case the cortical cells themselves are less diseased. There may be no physical symptoms beyond a general dwarfing of the entire body; on the other hand the patient may not be able to stand or walk, feed himself, speak, or indeed do anything save exist if cared for. Those of a higher grade are active, energetic, easily made angry, affectionate but wilful, having no power of continuous attention,—indeed resembling superficially, but only superficially, the monkey tribe. As a rule, they either cannot speak at all or only a few words. This is probably not a true aphasia, but due to their general low mental state. They are apt to be thieves, but to appropriate things useless or useful indiscriminately. Fortunately, the sexual instinct in the majority of cases is wanting. In some the organs of generation develop, but I know of only one case, that of Ireland, in which a microcephalic woman became pregnant. The child was born dead.

There is no treatment save to give what little education is possible. The surgical intervention (craniectomy) proposed by Lannelongue has fallen into disuse. It was based on a false pathology and had no good results.

Local Cerebral Defects.—As said above, a skull of normal size may contain an abnormally small brain. Besides this there may be an atrophy or a lack of development of some one part of the brain,—for example, one cerebral hemisphere. Most frequently this is caused by hemorrhage in the meninges at birth, but the disease process may occur during intrauterine life. Osler, for example, reports the case of a fœtus about six months old, in the left basal ganglia of which was a recent clot which had broken into the ventricle. The mother had died of typhoid fever. It is scarcely possible that any fœtus could survive till birth after suffering a cerebral hemorrhage, but it is very possible that many of these cases are caused by intrauterine obliterating vascular disease causing lack of growth. Whether any of them are really developmental defects is not yet determined. Clinically, atrophy of one hemisphere is a frequent cause of a congenital spastic hemiplegia. The paralyzed side is the seat of choreic or athetoid movements. Epileptiform convulsions are frequent. Frequently there is imbecility. Mills relates a case, which was not, however, intrauterine in origin, but followed convulsions at eighteen months, in which mirror-writing was present. This is met with now and again in both the intrauterine and birth hemiplegias. Hemianopsia or irregular contraction of the field of vision may occur. There is often no visual disturbance. In many of the cases in which the atrophy occurs during intrauterine life the skull is very asymmetrical, being of much smaller volume on the diseased side. This was so marked in one patient on whom a surgeon operated that the longitudinal sinus was displaced and he cut into it, causing an outflow of blood, and death.

There are still more localized defects of development or diseased areas. Thus the frontal lobes may be defective and the child, presenting no paralytic or sensory symptoms, will be mentally deficient. Peterson reports a case in which both occipital lobes and the motor area on the right side were atrophied. The patient was idiotic, epileptic, slightly hemiplegic, and could scarcely distinguish light from darkness.

Some cases of congenital cerebellar ataxia seem to be due to real defects in development, not to disease but to an error in development inherent in the embryo. Thus, Nonne describes a small cerebellum, without sclerosis and without degeneration, indeed normal in all regards except as to relative size. The spinal cord was also diminutive. Before Nonne, Marie had described a similar condition under the title "hereditary cerebellar ataxia." The symptoms are somewhat like those of Friedreich's ataxia. There is titubation, choreiform or ataxiform movements of the arms and legs, and increased knee-jerks. The ocular symptoms are important. There is nystagmus, diplopia, and sometimes amblyopia or color-blindness, or contraction of the visual fields. Optic nerve atrophy is characteristic. Other authors have described cases more or less resembling this type, and more or less departing from it. Indeed, there is clinically an unbroken chain of cases from Friedreich's ataxia on the one side to the so-called hereditary spastic palsy on the other. The difference in symptomatology must depend upon which part of the cerebrospinal axis is lacking in development. If the cerebellum alone be deficient the symptoms can not be the same as when the cord alone or both cord and cerebellum are involved. The symptoms of hereditary ataxia do not appear at birth but later, usually at about puberty.

Absence of the Corpus Callosum.—Nothing is definitely known of the functions of the corpus callosum except that it contains commissural fibres binding together the hemispheres. Bruce[1] states that it may be entirely absent without the patient showing any symptoms. As a rule, however, its absence is associated with other cerebral defects and accompanied by idiocy or imbecility.

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[1] Brain, vol. xii.

Abnormal Cerebral Fissuration.—Slight variation in the direction and number of the minor fissures of the brain is common. It has no pathological significance. Sometimes the variation from the normal is very marked and it has been attempted to set up a type of criminal, epileptic, and congenitally insane brain. Moritz Benedikt studied the matter for years and concluded that in either of these conditions certain cerebral stigmata are present. These are excessive development of principal and subordinate fissures and excessive confluence of fissures either by elongation of the principal fissures, or by their anastomoses through the great development of subordinate fissures connecting them. Benedikt's papers led to much discussion, which indeed still continues, and the question remains unanswered whether there is a type of brain which predestines its owner to become a criminal or an epileptic. In at least one form of insanity congenital in origin,—namely, paranoia,—there are sometimes marked deviations from the normal fissuration. Berkley says that in his experience it is the most striking thing on gross examination of the brain in that disease. Really no other gross lesion is ever present.

Cerebral Heterotopia.—Occasionally there is found on microscopic examination of a brain, usually one showing gross defects or abnormalities, small gray nodules or streaks in the white matter of the cerebrum or cerebellum due to the presence of tissue rich in nerve-cells and poor in medullated fibres. They are bits of gray matter out of place. Van Gieson has shown that in the spinal cord heterotopia is frequently an artefact, occurring while the cord is being hardened or cut and mounted. This is not always the case, however. Misplacement of tissue may occur in the brain just as it does in the abdominal organs. Its cause is not clear. It probably, however, is caused either by an abnormal development of nerve-fibres causing the detachment of a small bit of gray matter from its proper place, or by the primary development of gray matter in such a situation as to be later surrounded by white matter. Occasionally also anomalous bundles of white fibres are found in the medulla. Heterotopia never produces any symptoms.

Congenital Hydrocephalus.—External hydrocephalus is an accumulation of cerebrospinal fluid within the subarachnoid space. It is frequent in cerebral atrophy. In internal hydrocephalus the fluid is within the ventricles. It is caused by developmental defect of structure, intrauterine inflammation of the ependyma, and pathological conditions of the blood. It may occur in children of healthy parentage and whose mothers have passed through pregnancy without any illness. On the other hand, several children in a family may be affected. As a rule, but not always, the head shows enlargement at birth. Many children with this disease are born dead, some survive for a short time, a few, and if very slight cases are included, a quite large number, attain full maturity. The shape of the head is characteristic: the cranium is hemispherical or elipsoidal and the face appears relatively small. The bones of the vault are thin and separated. Many Wormian bones are present. The circumference of the skull varies greatly. It has been as much as forty inches. The amount of fluid varies greatly. The lateral ventricles are, of course, always dilated and their walls may be as thin as paper, the upper parts of the hemispheres being mere sacs. The cortex resists the pressure better than the white matter, but it, too, in a case at all severe, is decreased in thickness. The membranes, as a rule, are normal or slightly thinned. If the cause be inflammatory the choroid plexuses and the membrane lining the ventricles are thickened. The fontanelles bulge and pulsate with the heart-beat. If the child survive, many nervous symptoms develop. The mental symptoms vary from idiocy to high-grade imbecility. Some very slight cases present no symptoms at all except the deformity of the skull. Often there are cerebral palsies and epileptiform convulsions. There may be total blindness from atrophy of the optic nerves produced by direct pressure upon the chiasm and tracts. Optic neuritis is much less common. Strabismus is frequent. The diagnosis presents no difficulty. Treatment, either medical or surgical, has so far availed nothing.

Defective Development of the Cranial Nerve Nuclei.—Gowers describes under the title "infantile oculofacial palsy" a rare condition which may be present from birth or appear later, characterized by palsy of the external ocular muscles, paralysis of the face, and ptosis, but without involvement of the internal ocular muscles. He suggests that it is due to defective vital endurance in the nuclear structures. Sachs, using Schapringer's case as a text, shows that the motor branch of the fifth, the hypoglossal, the third, the sixth and seventh nerve nuclei may all be affected. On the other hand, only the nucleus of one facial nerve may be affected. I know of no post-mortem study of this condition, but it would seem to be caused by a true defect in development.

Congenital Absence of the Visual System.—This is a very rare condition. Absence of the visual system within the skull never occurs save in association with anophthalmos or microphthalmos. It is difficult to tell where to draw the line separating these two conditions. E. Treacher Collins and J. H. Parsons lay down the following rule: When there is complete failure of the essential nervous mechanism of the eyeball, anophthalmos is the proper term; when any of the nervous elements are present, no matter how imperfect, microphthalmos. Total absence of the eyeball is probably always a true developmental defect: microphthalmos is probably sometimes developmental, sometimes due to intrauterine disease. Zentmayer and Goldberg[2] describe very carefully the microscopic appearances in a case of microphthalmos. More interesting to neurologists, however, are the studies which have been made of absence of the visual system within the cranium. Quite a number of cases have been studied, but that of Spiller[3] is the best, and I shall quote largely from it. The patient was an idiot boy twenty-two years old. Spiller had regarded him in life as a case of cerebral spastic paraplegia with absence of the eyeballs. At autopsy the optic foramina were found to be absent. The orbital contents were not allowed to be removed and hence whether there was a rudimentary eye present could not be determined. Nothing resembling an eyeball was seen. No trace of optic nerves, chiasm, or optic tracts could be found. There was no sign of an external geniculate body on either side, and the thalamus on each side had nothing resembling an optic tract passing to it. The brain was small, firm, and not œdematous. The left ascending frontal convolution in the centre for the arm was very small. The occipital lobes and the cunei were very small. The calcarine fissure was short. The external arcuate fibres were unusually well developed. The spinal cord was small, but otherwise normal. On microscopic examination the cortex of the left calcarine fissure possibly contained fewer cells than normal. The optic radiations in the frontal sections of the occipital lobe were not entirely absent. Spiller draws the following conclusions: The chief "primary" optic centre is the external geniculate body. The pulvinar of the optic thalamus is also an important "primary" optic centre. The anterior colliculus of the quadrigeminal body in man has an unimportant relation to vision. The hypothalmic body, the habenula, and the internal geniculate body, probably are not parts of the visual system. The cortex of the calcarine fissure may contain nearly the normal number of cell bodies, even though the visual system may be undeveloped. The nerves to the ocular muscles and their nuclei may be developed, even though the visual system is absent. A condition such as this (See Chapter I, Figs. 1 to 4) is developmental and not caused by acquired disease, but as to the cause and mechanism of the developmental defect we know nothing. Nothing is known with any certainty as to any hereditary influence which might act as a predisposing cause. All the cases I know of, have occurred in people with more or less mental deficiency. It is sometimes associated with cleft palate or harelip. The majority of the patients die immediately or not very long after birth. Only a few reach maturity.

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[2] Annals of Ophthalmology, January, 1904.

[3] University of Pennsylvania Medical Bulletin, February, 1902.

Abnormalities in the Development of the Skull.—I shall consider only the defects of growth which cause congenital cranial hernia and the abnormalities which are associated with certain ocular disturbances. The microcephalic skull has already been described. *Cranial hernia* includes encephalocele (protrusion of the brain substance), hydrencephalocele (encephalocele containing fluid and with a cavity usually communicating with the ventricles), and meningocele (protrusion of the cerebral membranes).

Almost always the hernia occurs between bones and not through a hole in a bone. It almost always occurs in the median line, usually in the occipital, rarely in the frontal region. It very rarely is seen between the temporal and parietal bones. Cranial hernia produces a tumor under the scalp. An encephalocele is usually opaque and with a broad base. Meningocele and hydrencephalocele are translucent and pedunculated. It is justifiable to puncture with a hypodermic needle to determine whether the tumor is solid or contains cerebrospinal fluid. If the latter be present the tumor must be intracranial in origin and cannot be a vascular growth, a sebaceous cyst, or an abscess. All of these have been mistaken for cranial hernia. Frequently the opening in the bone can be felt, the mass may pulsate with the heart beat, and may increase in size as the child cries. Various other anatomic defects are apt to be present. Convulsions often occur. As a rule, the patients die after a very short time. In meningocele the sac has been successfully removed.

Congenital Deformity of the Skull associated with Optic Atrophy.—Post-neuritic optic atrophy associated with deformity of the skull has been reported in a small number of cases. The question is whether the deformity directly causes the disease of the nerves, whether both arise from a common cause, or whether the two conditions are coincidental. All authorities agree that there is a closer relation than coincidence. According to Norris and Oliver, vision is defective in greater or less degree from birth. Some are born blind, others have fair vision in early life, but slowly sight diminishes until, during adolescence, total amaurosis is reached. Some retain a little vision even in advanced life. Virchow and Hirshberg attribute the optic neuritis to some intracranial inflammatory trouble, in most cases probably a pachymeningitis,

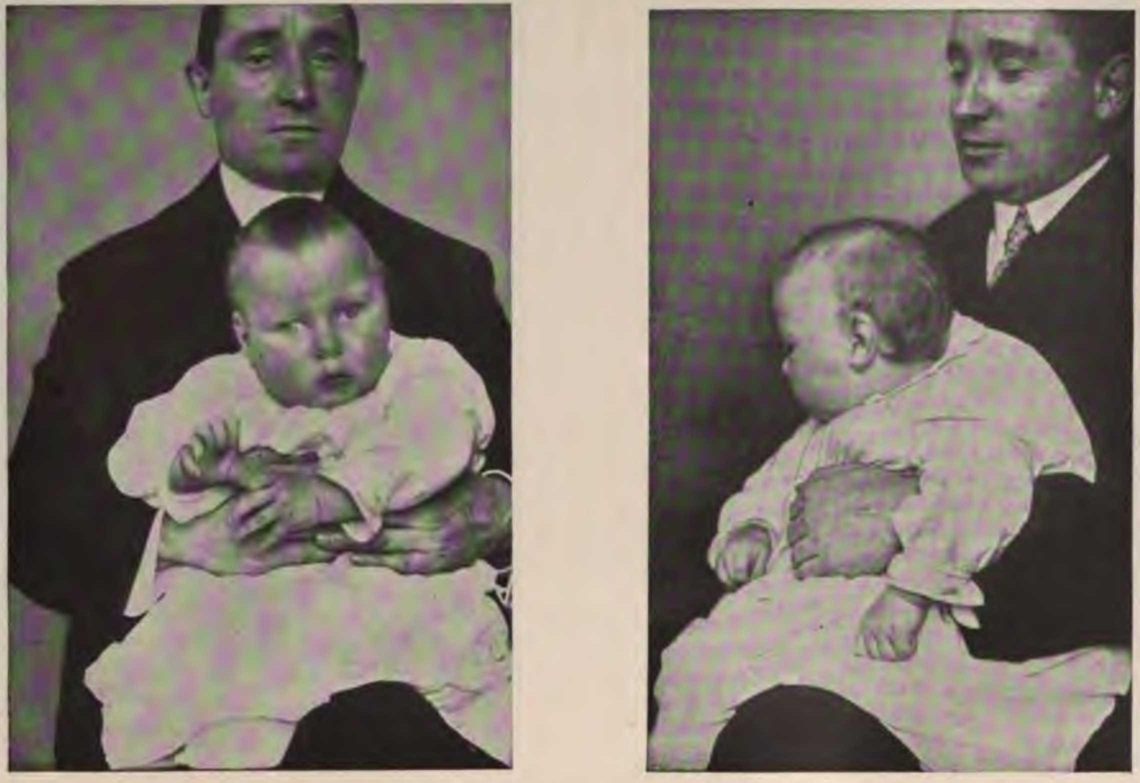

Fig. 1.—Case of Dr. Posey, with atrophy of both optic nerves due to malformation of skull.

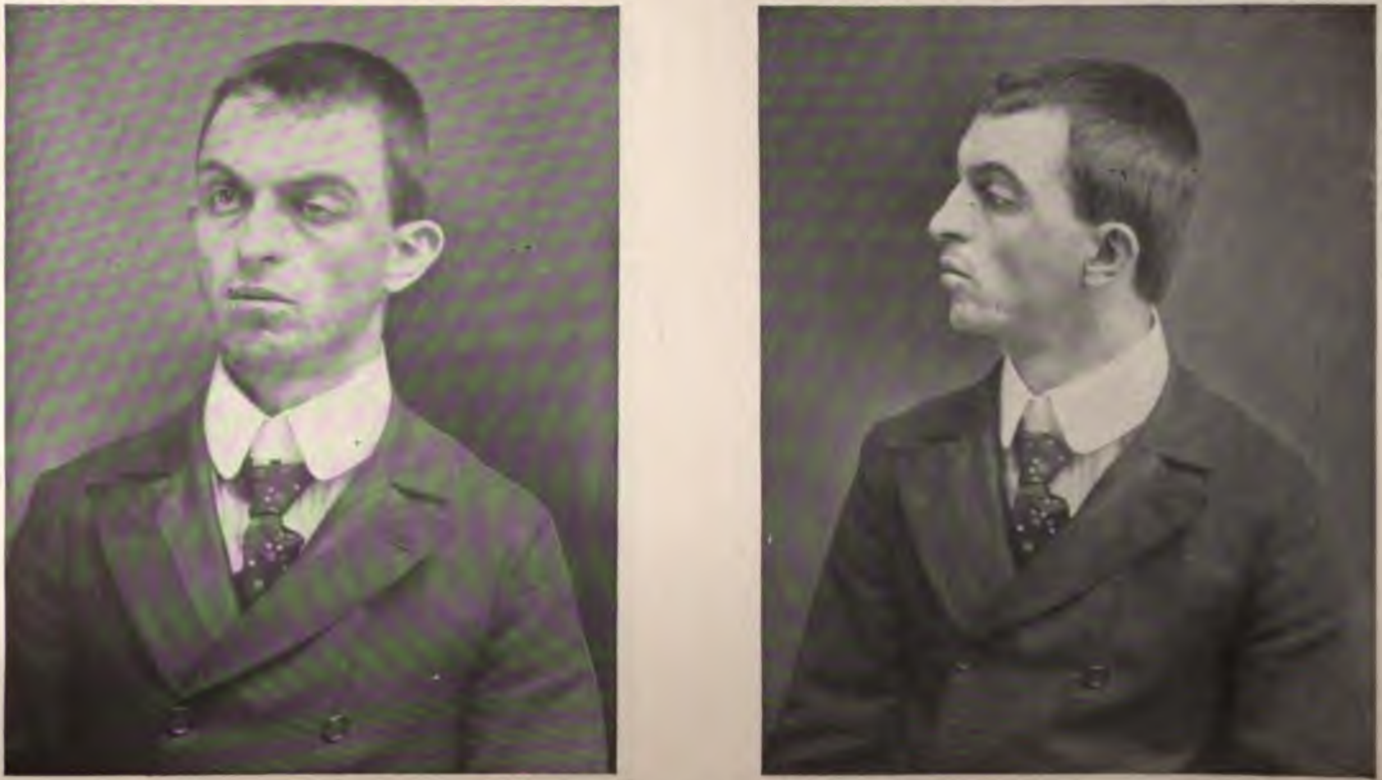

FIG. 2.—Case of Dr. Posey, showing ptosis of left eye in a subject with malformed skull.

which may also cause the cranial deformity. Friedenwald[4] believes the condition is caused by increased intracranial pressure the result of premature synostosis of the cranial bones. Opposed to this is the fact that in many cases of microcephalus there is no disease of the optic nerve. Necropsies, of which there have been three, throw some light on the causation. In Michel's case there was hyperostosis of the skull, narrowing of the optic foramina, and thickening of the meninges; in Manz's the base of the skull was very irregular and the optic foramina were narrow; in Ponfick's the optic foramina were small. In the first two cases the skulls were oxycephalic (steeple-shaped); the deformity in the third I do not know. In consequence of these post-mortem findings, Weiss and Brugger examined four steeple-shaped skulls in the Heidelberg Institute. They found the optic foramina of normal width, but the orbital cavities reduced in size, being very shallow, and the alteration in shape being caused by the greater wing of the sphenoid forming the posterior instead of the lateral wall of the orbit. The dorsum sellæ turcicæ was enlarged and pushed forward, which they suggest might have caused pressure upon the chiasm and optic nerves. Friedenwald, in discussing their paper, points out that such pressure would cause a simple, not a neuritic atrophy. Deformities other than oxycephaly may be associated with optic atrophy. In Friedenwald's table of nineteen cases, twelve were oxycephalic, three long skulls, one leptocephalic, one with broad forehead (synostosis of the sagittal suture), one with a bulging temporal region, and one in which the deformity is not described. Oliver has published an elaborate paper on "Blindness from Congenital Malformation of the Skull."[5]

Oliver also points out that blindness may occur in association with deformity of the skull without any disease of the optic nerve. He reported the following case.[6] The patient was a four-year-old girl who, the mother stated, could see a little till her third year. She showed no ophthalmoscopic sign of disease. The eyes were prominent. The head was disproportionately small and quite deformed posteriorly. The occipital protuberance was almost wanting. The occipitoparietal suture was not well closed, the thickened and serrated edges of the occipital and parietal bones curving outwardly. Her mental condition was good. He believes the blindness was cortical in origin, and suggests as a possible cause "stretching and inflammatory processes taking place in and around a series of imperfectly made and quite readily disturbed tissues in the occipital region." The most frequent cause of the atrophy is surely that described by Virchow and Hirshberg.

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[4] American Journal of the Medical Sciences, May, 1893; Archives of Ophthalmology, 1901.

[5] Proceedings of the American Philosophical Society, vol. xli., No. 169.

[6] American Journal of the Medical Sciences, January, 1902.

PROGRESSIVE FACIAL HEMIATROPHY.

This affection is very rare. It is most apt to begin before the tenth year, is less frequent in adolescence and very rare in old age. In 92 cases collected by Hermann Steinert, 60 cases occurred in females, 30 in males, and in two the sex was not mentioned. It has been reported twice in parent and child. Its true causes are unknown. The most frequent preceding event which could stand in a causal relation is trauma of the face. It has followed the acute infectious fevers after a longer or shorter period, and has occurred during the course of many nervous diseases,—*e.g.*, hysteria, multiple sclerosis, locomotor ataxia, epilepsy, and syringomyelia. Its association with none of these diseases, however, has been more frequent than can be accounted for by the law of coincidence. There seems to be a relationship between it and scleroderma. The two diseases occur together with relative frequency. Facial neuralgia so often precedes the appearance of the atrophy that it may properly be regarded as a symptom rather than an associated disease.

Little is known as to its morbid anatomy and the real seat of disease. Mendel had one autopsy. The patient had been ill twenty-five years. Along with atrophy of the face there was atrophy of the left arm. Postmortem examination revealed proliferating interstitial neuritis of the left trifacial, most marked in the second branch. The facial was normal, but the left musculospiral was affected in the same way as the trifacial. In an atypical case reported by Homén there was a tumor pressing upon the Gasserian ganglion and the branches of the trifacial. There was not only facial hemiatrophy but also anæsthesia, palsy of the oculo-motor nerve, and atrophy of the tongue. There was degeneration of all branches of the trifacial nerve, and of the third, fourth, sixth, nd seventh nerves. Hemiatrophy may also follow injury to the fifth . Some authors tend to the view that the seat of the disease is in vical sympathetic. Thus Seeligmuller reports a case following in that region. Certain authors regard it as a true skin disease. surely an error. It is a disease of nutrition and most likely upon perversion, not mere abolition, of function in the fifth the sympathetic system, or both. Congenital predisposition be important for its occurrence,—at least it is most frequent uropathic.

Symptomatology.—The first objective sign of disease appears in the skin. A circumscribed spot on the side of the face becomes dead white or yellowish and at the same time the skin grows thin. Rarely two spots are attacked at once. The disease is most apt to commence in the orbital region or the lower jaw. Soon there is a loss of fatty tissue under the skin and a shallow pit is formed. The affected area extends and involves not only the skin but the subcutaneous tissues and bone, and in less degree the muscles. There is never reaction of degeneration of the muscles nor complete palsy. The fatty tissue of the orbit disappears and the eyeball sinks in. The final color of the skin is a shallow yellow or brown. The brow and beard fall out or become white. The hair of the head is rarely affected. Perspiration may be increased, but the secretion of the sebaceous glands lessens or ceases. The disease is progressive, but in the greater number of cases ceases at the middle line. It may, however, pass over to the other side. The neck muscles and the arm of the same side may be involved. Except at the beginning its progress is very slow, and though recovery never occurs,—*i.e.*, the face never regains its natural aspect and form,—the process may cease at any time. It may recur. The deformity in an advanced case is marked and characteristic. The diseased side is depressed below the level of the other from the forehead to the chin. The line of demarcation is clear-cut.

There are many minor symptoms. Pain or itching in the affected region is frequent. The tongue, hard and soft palate, and the gums may atrophy. The teeth may fall out. There is never complete anæsthesia, but there may be hyperæsthesia. The atrophied skin may feel to the patient tense and mask like. Rarely the pupil is dilated or very small. There is never disturbance of vision, but taste and hearing may be impaired. In very few cases neuroparalytic ophthalmia has been observed. Sachs and other authors refer to cases in which there were marked spasm of the masseters, clonic, tonic, or both.

The disease does not shorten life. The only conditions with which it can be confounded are congenital facial asymmetry, facial palsy, and facial hemihypertrophy of the opposite side. These need only to be remembered to avoid error. It may be associated with scleroderma, but the differentiation is not difficult. Scattered through the literature are reports of atrophy occurring in other parts of the body, similar in onset and progress and involving the same tissues. It is probable that some of these at least are identical pathologically with this disease.

Treatment has no influence.

PROGRESSIVE FACIAL HEMIHYPERTROPHY.

This condition is still rarer than hemiatrophy. It is characterised by an overgrowth of one side of the face. The soft parts may be alone involved or together with the bones. It usually is congenital, but may first appear in childhood or youth. Its causation and morbid anatomy are unknown. The earliest case I know of was reported by Stilling in 1840. The hypertrophy followed an injury which occurred when the patient was nine years old. Montgomery's case followed two years after an abscess of the cheek. It has followed various illnesses and without any apparent cause. The cases reported are too few to draw any conclusions from as to causation. There may be no sign of disease beyond the mere increase in size. Only the soft parts with or without the mucous membrane of the mouth may be affected, or the bones, or one or more of them, may be affected also. Only a part of the face may be involved, as the cheek. In Hoffman's case,[7] that of a girl of fourteen years, in whom the change was first noticed at two years, there was a growth of hair on the upper lip. The skin may be normal or thickened and pigmented. Once only has it been recorded as thick and coarse. It rarely is hyperæmic. There are no characteristic sensory symptoms. Berger's case, however, followed an old trifacial neuralgia. Dana's case occurred in a young giant. The sebaceous and sweat glands may be overactive. There may even be an increase of saliva on the affected side. There are no ocular defects nor disturbances of vision. The only changes found so far at autopsy have been hyperostosis, uniform or in masses, an increase in the adipose tissue, and an atrophy of muscle fibres. It is progressive during youth but ceases, or almost ceases, at maturity. It never causes death. Once, by pressure of the thickened skull upon the brain, it caused epileptiform convulsions.

OTHER PROGRESSIVE HYPERTROPHIES.

Occasionally there is seen an hypertrophy of one entire half of the body, or of one arm or leg, or finger, or as is reported in one case, one side of the face and the corresponding arm and the leg of the opposite side. The symptoms are precisely the same as in the facial form and it is probable that the pathology is the same. It seems, however, that the latter is more frequently acquired than the former. All the tissues of the part are affected, the skin, subcutaneous soft parts, the muscles and the bones. Arnheim indeed describes a case in which at autopsy the internal organs were found hypertrophied. The right ovary, kidney, lymphatic glands, and even the right side of the heart were enlarged. Hymanson[8] found by actual measurement that in his case growth was much more rapid on the affected side. We know nothing about the cause of the condition, but Anderson gives the following views as to its etiology: (1) A congenital lesion of the vasomotor centres leading to vascular stasis, (2) a primitive vice of the middle lamina of the blastodermic membrane, (3) partial intrauterine strangulation of the affected member, (4) an inherent tendency of the tissues to appropriate an excess of nutriment. England found at autopsy on a child, in whom the left side of the face and head was hypertrophied, neurofibromata involving all the cranial nerves.

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[7] Deutsche Zeitschrift für Nervenheilkunde, vol. xxiv. p. 425.

ACROMEGALY.

Acromegaly was first described by Pierre Marie in 1886. Long before that time many writers had reported cases of curious deformity and strange alteration in physiognomy coming on in youth or adult life, and of course giantism was well known; but Marie was the first to separate from these cases of acquired abnormality of anatomic structure the type which we now call acromegaly and to show that it is a distinct disease with a definite symptomatology and probably due to a single cause. It is of interest to oculists because of the ocular symptoms due to the associated, if not causative disease of the hypophysis, and to neurologists because of the obscurity of its pathology and the occurrence of nervous symptoms.

The most striking feature of the disease, that which makes it possible to diagnose a well developed case at sight, is the alteration in physiognomy resulting from an overgrowth of the hands and feet, the head, jaws, lips, and tongue and a cervico-dorsal spinal curvature. The most frequent first subjective symptom is headache, dull or sharp, local or general, paroxysmal or lasting. The change in bulk is first called to the notice of the patient by the fact that he must wear larger and larger shoes or gloves. Even before this, friends may observe an inexplicable alteration in the facial expression.

Causation.—The predisposing causes are unknown. Direct inheritance seems to play no part, though a few cases have been reported in brothers. It is possible that in them pathological incidents during intrauterine life acted as predisposing causes rather than heredity. Most frequently the cases are isolated, one in a family. The influence of race is unknown, but it seems to be very rare in the American negro,

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[8] Archives of Pediatrics, vol. xx. p. 428.

Berkley alone, so far as I know, having reported a case. According to the tables of Hinsdale, males are more predisposed than females; according to Dana, the two sexes are affected with about equal frequency. The period of greatest frequency of onset, so far as that can be determined in a disease so insidious in its development, is from the twentieth to the thirtieth year. A few cases have been reported in which the onset occurred after the seventieth year and recently there have been published instances in which the disease was alleged to be congenital. It is by no means certain that these are genuine or that they should not be classed elsewhere. They may bear the same relation to the acquired disease that infantile cretinism bears to myxœdema. The relation of giantism and acromegaly is much discussed. They certainly are not the same thing and the latter is not infrequent in the former.

The exciting causes are also unknown. The rule is for the disease to develop without the previous occurrence of anything which could be assumed to be a cause. It may follow an acute infectious fever, rheumatism, trauma, childbirth, prolonged emotional strain and, indeed, any disease. It sometimes occurs in imbeciles and epileptics. One thing is sure, alcohol and syphilis are not exciting causes. If they were it would be much more common. I shall discuss the relation between disease of the hypophysis and acromegaly under the heading, *morbid anatomy.*

Symptomatology.—The usual picture is as follows: Some time between late adolescence and middle life the hands and feet begin to enlarge and the face to alter. At the same time headache begins, is much treated and persists. The lips and eyelids become thickened, the ears enlarged, the nose broadened, lengthened, and the septum thickened. The mucous membranes hypertrophy and the tongue becomes unwieldy from its increased bulk. While these changes are occurring in the superficial tissues, similar ones take place in the bones of the skull and especially in the face. The lower jaw enlarges so much as to cause prognathism, the line of its circumference extending far beyond the upper jaw. The cheek bones become prominent. The voice grows hoarse and deep. The skull increases only moderately in circumference, but the fingers lengthen and broaden and become like spades. Visual disturbances appear, contraction of the fields or hemianopsia. The patient becomes dull, lethargic, stupid, unfit for any work. A true melancholia may appear. In the meanwhile the vertebræ have become affected and cervicodorsal kyphosis sets in. Finally the patient dies from the disease of the hypophysis, with symptoms of brain tumor or from some intercurrent affection. The duration of the disease

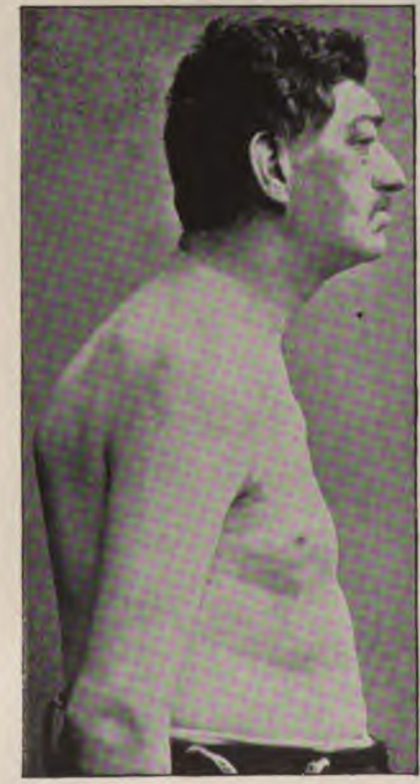


FIG. 3.—Acromegaly, showing facial deformity and marked dorso-cervical kyphosis. (Case of Dr. F. A. Packard. American Journal of the Medical Sciences, Jan.-June, 1892. For photograph of brain from this case, see chapter IX, page 428).

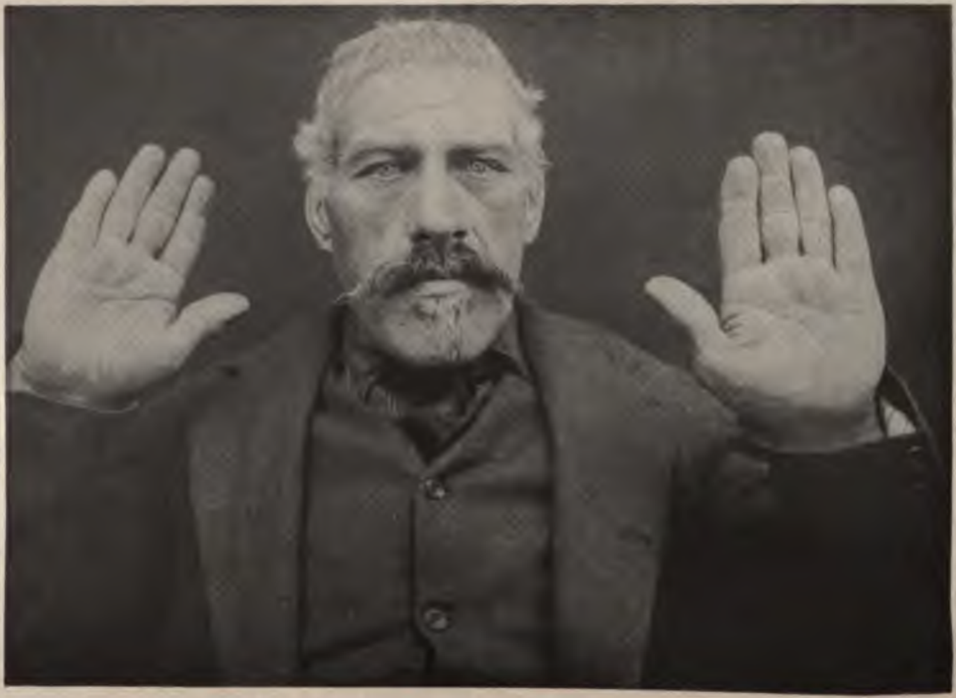


FIG. 4.—Acromegaly, showing deformity of the face and hands. (From International Clinics.)

is very variable, and using this as a means of classification Sternberg divides it into three varieties, namely, the benign, which lasts about fifty years and in which the changes are very slight, the usual chronic form having a duration of from eight to thirty years, and an acute malignant form, in which death usually occurs within four years. In the last the hypophysis is always sarcomatous. Notwithstanding the high mortality of the disease and its tendency to slowly but surely progress, in some cases the pathologic process ceases and in that sense the patient recovers. The lesions already produced of course remain.

Even at the expense of a little repetition the symptoms, both objective and subjective, deserve individual study. The objective symptoms are the more important in diagnosis. Most striking is the change in the general appearance of the patient, caused by the thickening of the cutaneous, subcutaneous, and in less degree, the bony structures of the hands, feet and skull. As a rule, the enlargement begins simultaneously in all parts, but occasionally one extremity is affected before the others. The fingers and toes enlarge more in circumference than in length. They become sausage-shaped, and the broadened hands spadelike. The disproportion between the much enlarged hands and the normal arms and forearms is so marked as to be grotesque. On the palms and soles the epidermis and true skin is much thickened and pads form. The nails are flattened and striated longitudinally. The orbital ridges and the external occipital protuberance are marked. The increase in the circumference of the skull is largely due to enlargement of the sinuses, but sometimes the cranial bones are thickened.

The bony changes in the thorax are characteristic. As stated above, cervicodorsal kyphosis is common,—sometimes it is so marked that the ribs touch the brim of the pelvis. The sternum is thickened and widened, the ensiform being very prominent. The increased thickness of the manubrium is important to remember, because the percussion note which it gives may be supposed to be due to a persisting thymus gland beneath it. Erb regards sternal dulness as pointing strongly to the presence of the thymus gland. This sign is surely not infallible. The clavicles and ribs are massive. On account of the changes in the bones of the thorax its shape is much altered. Its anteroposterior diameter becomes proportionally greater than the lateral. The pelvic bones and the great joints of the body are not affected. Except in the so-called giant type of the disease the long bones remain normal. The neck is thickened and the abdominal walls are sometimes pendulous.

The ocular symptoms are important. The eyeballs are often prominent and there may be extreme exophthalmos, due probably to an increase of the fatty tissue behind the bulb. Hinsdale quotes Burchard as reporting one case in which the eyeball itself was enlarged. Rarely severe ocular neuralgia occurs. Nystagmus is sometimes seen. There may be partial or complete palsy of the third and sixth nerves. The irides may not react to light at all or hemianopic iritic rigidity may be present. Some visual disturbance always exists. There may be only slight dimness of vision or absolute blindness consequent upon optic atrophy due to papillitis caused by enlargement or tumor of the hypophysis. The form and color fields may be concentrically contracted or there may be true hemianopsia. Disease of the hypophysis and possibly, as Broca believes, bony changes in the optic foramina consequent upon the overgrowth of the lesser wing of the sphenoid bone may produce bitemporal hemianopsia,—*i.e.*, blindness in the nasal half of each retina. Homonymous hemianopsia has been described by de Schweinitz, among others. The type of hemianopsia present depends upon the direction of the pressure upon the optic chiasm and optic nerves. Sometimes there is total blindness in one eye and hemianopsia in the other. The ophthalmoscopic picture is the same as is found in tumor of the brain in any region.

The larynx is enlarged in all directions, the mucous membrane and the cartilages thickened, making the voice deep and hoarse. The thyroid may be enlarged or diminished, hypertrophied or sclerosed. Very rarely Graves's disease occurs. Menstruation in women and sexual desire and power in men are apt to disappear early. At first muscular power is retained or even increased, but later there is marked weakness. The knee-jerk is usually normal. It may on account of complications be absent, increased, or unequal on the two sides. Electric conductivity is increased. The urine frequently contains albumin and glucose and may be enormously increased in quantity. The blood presents no noteworthy changes. Disturbances of sensibility do not occur in uncomplicated cases, but hysterical hemianæsthesia has been noted. Extreme hunger and thirst are not uncommon. There is often cardiac enlargement with dilatation causing paroxysmal palpitation. The characteristic mental state is one of apathy, lack of energy, and marked sleepiness. There is almost always some emotional depression which may pass into a true melancholia. Some patients become simply a little silly, mildly demented, and unduly cheerful and good natured.

Morbid Anatomy and Pathology.—The skin, subcutaneous tissues, and the bones in the affected regions of course always show marked signs of disease. There is a true hypertrophy of the skin and the sweat and sebaceous glands, of the arterial walls, and of the sheaths of the

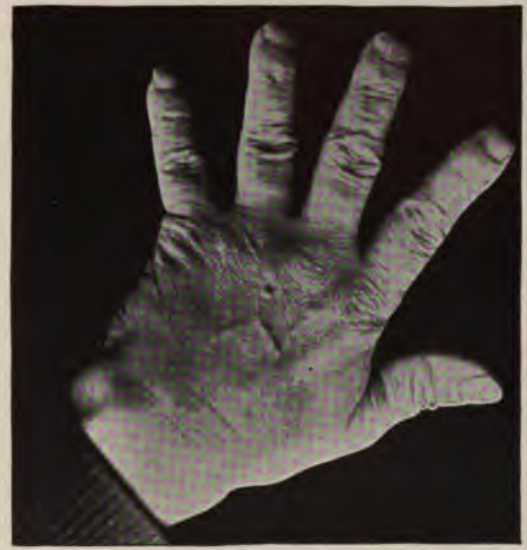


FIG. 5.—Showing deformity of hand. (Case of Dr. F. A. Packard.)

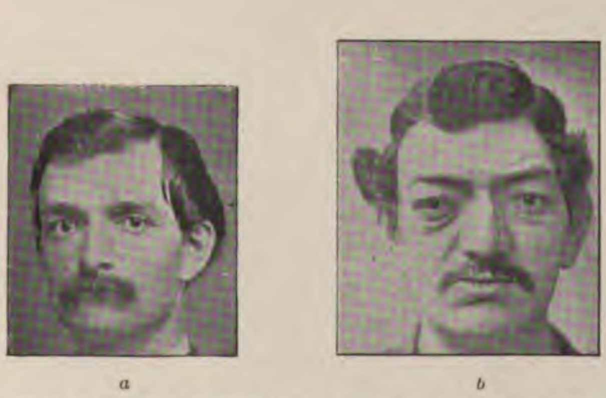


FIG. 6.—*a*. Photograph of the patient at twenty-two years of age. *b*. Photograph at forty-five years of age. (Case of Dr. F. A. Packard.)

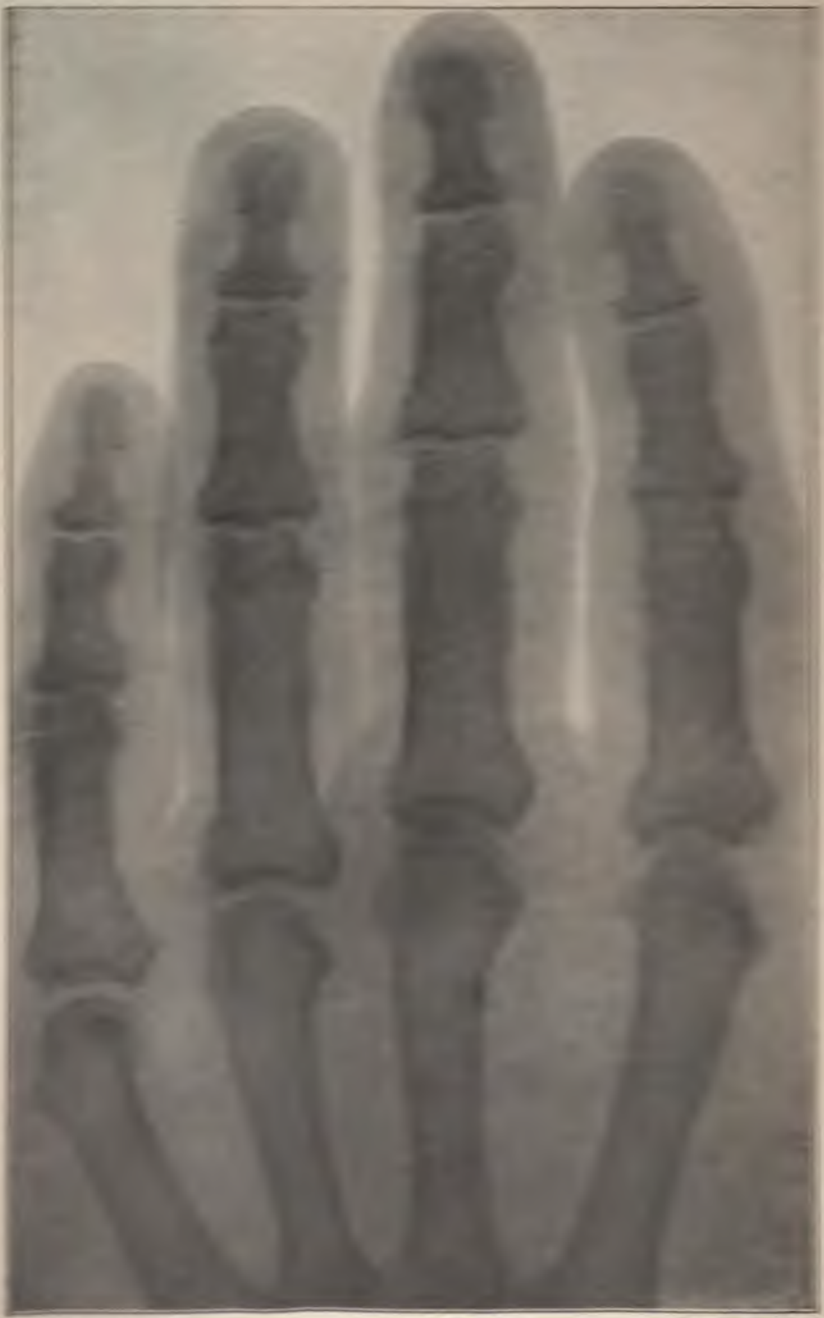


FIG. 7.—Portion of skiagraph of left hand of Dr. Walton's case of acromegaly. (Actual size.)

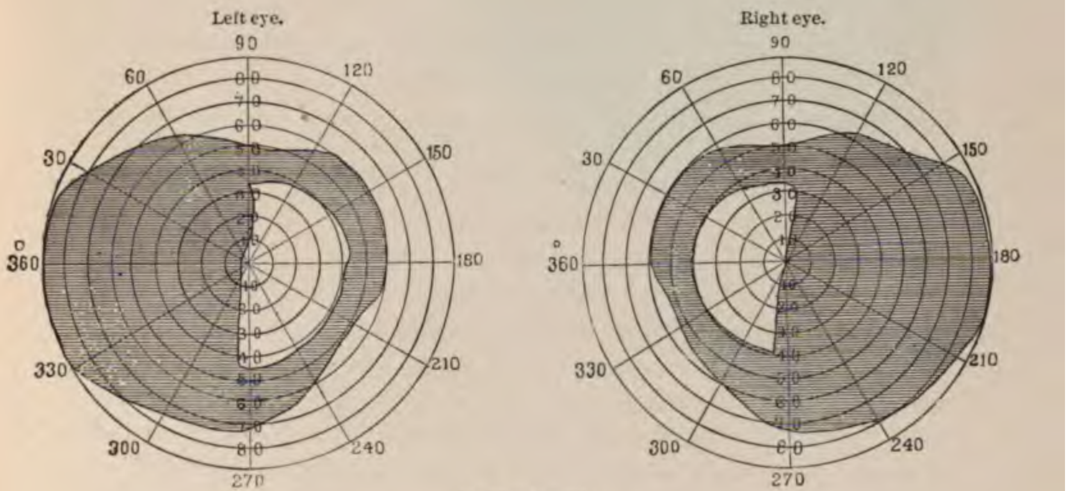
cutaneous nerves. In the bones the periosteum is thickened, the grooves of the blood-vessels deepened and enlarged, and in the skull and spine small extoses are present. The bones are thickened, and since this does not involve the whole bone symmetrically, deformity ensues. The lower jaw is always most affected. In the spine the vertebræ are ankylosed by bony overgrowth. The smaller joints present appearances similar to those seen in arthritis deformans. In a case of Broadbent's, X-ray examination showed that there was enlargement of the soft parts only, the bones being of normal size. This may mean, however, not that disease of the bone is not an inherent part of acromegaly, but that in this case the skin became affected first, as indeed may be the rule.

The organ most discussed in all studies on acromegaly is the hypophysis. It is more frequently found diseased than any other organ in the body, and not a few authorities hold that if it be normal acromegaly cannot occur. They explain the few cases in which normal hypophyses have been found at necropsy by claiming errors in clinical diagnosis. The following seem to be the facts in the matter. The hypophysis is diseased in the great majority of cases if not in all. It may be hypertrophied or the seat of a tumor. On the other hand, disease of it completely destroying its glandular structure and, of course, also its rudimentary nervous structure, may occur without any symptom of acromegaly appearing. Such disease may occur at any period of life and last for quite a long time. For example, a patient of mine who had adiposis dolorosa showed at necropsy a tumor as large as a walnut. Her illness had lasted many months. The kind of disease affecting the hypophysis seems to be a matter of no moment since hypertrophy and tumors of many kinds occur. Some writers hold that simple hypertrophy of the hypophysis is only a symptom of the disease, and not causative. Experimental destruction of it in lower animals does not produce acromegaly. This may, however, be due to the fact that death is apt to follow very quickly, that the hypophysis may have other functions in lower animals than in man, and that destruction of function need not, indeed ought not, to cause the same symptoms as perversion of function, such as may occur in a diseased but not totally destroyed organ. Altogether it is best to confess ignorance and admit that the true relation between acromegaly and hypophyseal disease remains unexplained. Others of the so-called ductless glands are often found diseased, notably the thyroid. The thymus, which should not be discoverable in an adult, is often present and the seat of a tumor. The ovaries and testicles are often atrophied. Slight and probably coincidental changes in the spinal cord, degeneration of the posterior and lateral tracts have been found.

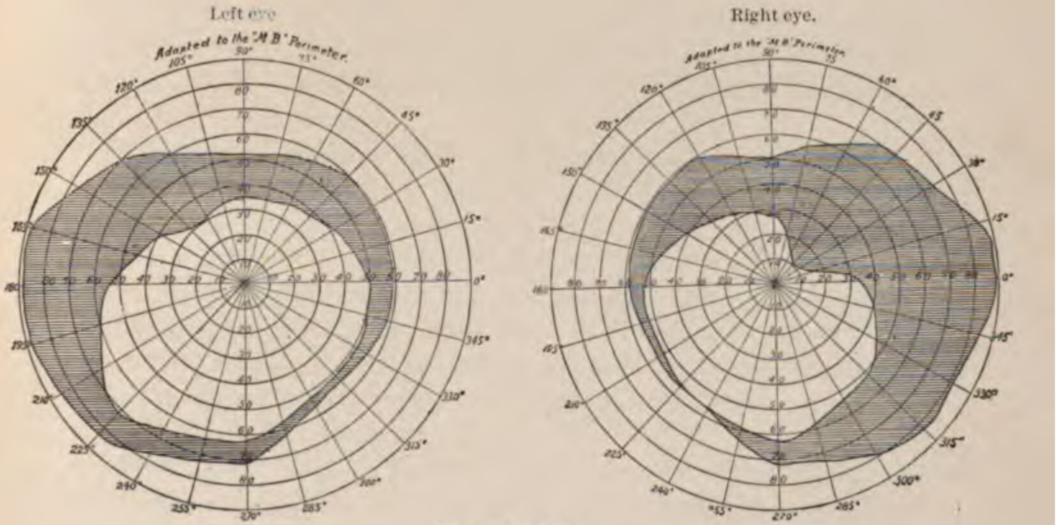
The spinal and sympathetic ganglia have, when examined, shown hypertrophy. Syringomyelia has been reported, but surely cannot have any causative influence. A few investigators have endeavored to throw light on the pathology of acromegaly by chemical studies, but so far with little success. Edsall and Miller, for example, made a very careful study of the urine in two cases, but could only show that there is "a growth of abnormal bone rather than a mere abnormal growth of bone, and that there are very marked abnormalities" [chemical] "in the soft tissues." All explanations of acromegaly heretofore offered have been purely hypothetical, except that of its hypophyseal origin which, while not proven, certainly has evidence in its favor.

Differential Diagnosis.—A fully developed typical case of acromegaly can be diagnosed at sight. There are, however, several diseases which may in fact be pathologically nearly related to it and which may be mistaken for it unless a careful examination be made. Myxœdema, for example, has a slight superficial resemblance to it, but the myxœdematous swelling involving the entire body, the local tumefactions particularly in the suprascapular region, the puffed face with obliteration of the lines of expression, and the brittle, thin hair are points quite sufficient to serve for diagnosis. Many giants are acromegalic, but in simple giantism there is symmetrical overgrowth of the entire body and not of the extremities alone. So-called partial giantism, hypertrophy of individual extremities, is recognized by its limitation to one part. In osteitis deformans (Paget's disease) the shafts of the long bones are chiefly affected, and in the head the bones of the cranium, not those of the face. In Paget's disease the face is triangular with the base up, in acromegaly it is egg-shaped. In Paget's disease also changes in the soft tissues are not present and the symptoms rarely appear before the fiftieth year. In leontiasis ossea the cranial bones alone are affected. Adiposis dolorosa is distinguished by the local swellings, the pain, the absence of deformity, and of ocular symptoms. The most difficult differential diagnosis of all is that from pulmonary hypertrophic osteoarthropathy. This disease also was discovered by Marie. In it the bones are more affected than the soft parts. The wrists are greatly deformed while the lower jaw escapes. Kyphosis is rare, scoliosis frequent. The hands are paw-shaped. There is always some pulmonary affection. X-ray examination is useful in diagnosis.

Treatment.—Nothing has been found which influences the course of the disease. Thyroid and hypophyseal extracts have no effect. Attempts to remove the tumor of the hypophysis have been followed by death in a short time.



Observed in 1885.



Observed in 1892.

FIG. 8.—Visual fields. (Dr. F. A. Packard's case of acromegaly.)

CHAPTER XV.

DISEASES OF THE SPINAL CORD AND SPINAL NERVES.

BY T. H. WEISENBURG, M. D.

MYELITIS.

MYELITIS is inflammation of the substance of the spinal cord. We have the acute, subacute, and chronic forms, depending upon the mode of the onset. When the inflammation involves the whole thickness of the cord for one or more segments, it is called transverse myelitis; if more extensive in continuous areas, we have the diffuse type; and if the process is at various levels, it is called a disseminated myelitis.

Etiology.—The exact cause of acute myelitis is not known, but it is believed to be an acute infectious disease due to a specific germ. In several instances staphylococci and streptococci have been found in the cerebrospinal fluid, and the pneumococcus has been found in a case of disseminated myelitis. Exposure to cold and wet, overexertion, and the acute infectious diseases, such as smallpox, diphtheria, typhoid fever, pneumonia, and gonorrhœa, some of the chronic blood conditions, and even gout, may cause myelitis, while syphilis is one of the most frequent causes.

Pathology.—Microscopically, the appearance depends upon the stage of the disease. In the acute period the blood-vessels are dilated and filled with red and white corpuscles. Around the vessels are found leucocytes and accumulations of round nuclei, and occasionally hemorrhages are seen. The nerve-cells are in various stages of disintegration. The axis cylinders are swollen and their myelin sheaths destroyed. The neurogliar fibrils are swollen, and their nuclei are much increased in number. Granular cells are found in abundance in the neurogliar tissue and around the blood-vessels.

Symptoms.—The symptoms of acute or subacute myelitis, with which we are concerned, depend upon the part of the spinal cord affected. Usually the dorsal cord is most intensely involved. The symptoms may come on rapidly, sometimes in a few hours, or may come on gradually, lasting for some weeks or months. There may be a feeling of malaise, and even a rise of temperature, and the patient may complain of a feeling of numbness or of a tingling sensation in the limbs, sometimes, also, of a pain in the back. Following this, or perhaps without these pains, there is a feeling of weakness in the extremities, which is rapidly followed by complete flaccid paralysis. At times a total paralysis comes on within a few hours. Sensation in all forms is much affected, the upper limit of the anæsthesia depending upon the location of the lesion. The reflexes may be lost at first; but in a short time the limbs will become spastic, the reflexes, such as the patellar and the Achilles jerks will be much increased, and the Babinski phenomenon is obtained. The vesical and rectal sphincters are involved from the beginning, there being at first retention of urine and constipation, followed by incontinence. Trophic disturbances, such as bed-sores, soon appear. Wasting of muscles and the reactions of degeneration are obtained, provided the cells of the anterior horns supplying these parts are destroyed. Lumbar myelitis causes somewhat different symptoms.

If the infection is intense, the disease extends rapidly, soon affecting the cells supplying the respiratory muscles and causing death. If the patient survives, the course of the symptoms will depend upon the severity of the lesion. Usually sensation will return before motion, and the patient will gradually show the symptoms of spastic paraplegia.

Ocular Symptoms.—Inflammation of the optic nerve accompanying myelitis is rare. Steffan and Erb,[1] in 1876, first described such an occurrence. Katz,[2] in 1896, collected twenty-one cases. Since that time seventeen have been recorded, to which the author is able to add one case of his own. This makes a total of thirty-nine cases; and when the large number of instances of myelitis are considered, the rarity of the affection is shown. (See Chapter VIII, p. 375).

In the majority of instances the disease first manifests itself in a rapid diminution of vision, mostly in both eyes, but sometimes only in one. Often some time elapses before the second eye is affected, which is frequently not until the first is on the way to recovery.

The diminution of vision is usually rapid. In one case, after twenty-four hours there was complete amaurosis. In other instances a few days, and sometimes a number of months, elapse before there is complete loss of vision. In the author's case one month passed before there was complete blindness.

It is not always necessary to have subjective symptoms of diminution of vision, for in a number of reported cases the patients did not complain of such phenomena, and even the ophthalmoscopic examination may be negative, the diagnosis of optic neuritis being made by the microscope.

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[1] Arch. f. Psych. u. Nervenkr., vol. x, No. 1.

[2] Arch. f. Ophthalm., vol. 42.

Usually the symptoms are ushered in by pains in the orbit and in the frontal region of the affected side. They are increased by movement of the eyeball, and there is sometimes acute pain on pressure. These pains have been variously explained. Some authors believe that they are due to an inflammation of the optic nerve sheaths or to a periostitis of the optic foramen. They have also been attributed to that form of inflammation of the optic nerve known as retrobulbar neuritis. Most probably they are due to the irritation of the sensory branch of the fifth nerve, which supplies the optic sheath. (See Chapter XVIII, p. 743.)

In the majority of instances the optic neuritis precedes the myelitis, sometimes by a few days, or from three to five months. The symptoms may come on together, or the optic neuritis may follow the myelitis, as in the author's case, after three months.

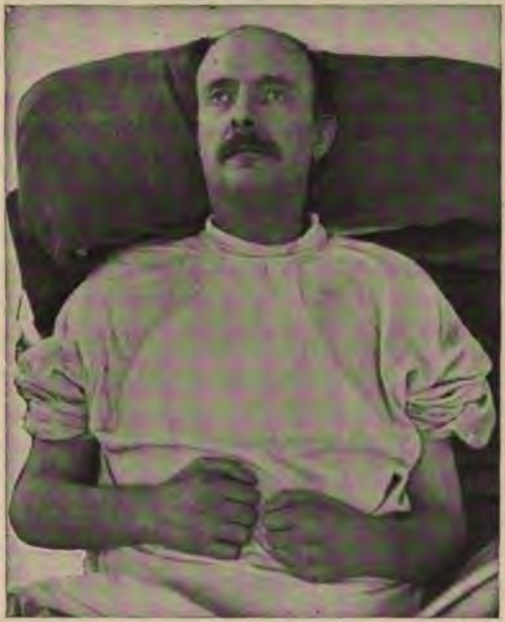

FIG. 1.—Case of meningo-myelitis with bilateral optic atrophy secondary to optic neuritis. Position of hands shows extent of power of elevation.

The condition of the pupils varies. Usually they are wider than normal and the reaction to light is lost. This has been explained by the existing amaurosis. The pupils, however, may be unequal, or one or both may be myotic. If the myelitis involves the cervical cord, especially the eighth cervical and first dorsal segments, there may be a contraction of the pupil, narrowing of the palpebral fissure, and enophthalmus, due to paralysis of the cervical sympathetic. Irritation of the sympathetic produces widening of the palpebral fissure, exophthalmos, and dilation of the pupil—as in one case (Bielchowsky), in which exophthalmos and swelling of the lids were seen. Nystagmus has been mentioned but once, by Brissaud and Brecy;[3] it being vertical in direction. Except in one instance, in which there was redness, nothing pathological has been found in the external part of the eye.

Disturbances of the fields of vision and of color-sense are not numerous, because the blindness is usually rapid and in all parts of the field. Central scotoma, a concentrically contracted field of one eye, or a quantitative light disturbance in a part of the field, have been described. Disturbances of color-perception have been mentioned a number of times, as well in the beginning as in the end of the disease. In one instance a unilateral color-blindness was found after the optic neuritis had disappeared.

Involvement of the other cranial nerves is rare. Mention is made of it in only four cases. The facial nerve was involved in one instance, while in another both internal recti muscles and the seventh and twelfth nerves were implicated. In the other two cases there was paralysis of both internal and external recti muscles, ptosis, and paresis of the facial and hypoglossal nerves. In several reported cases some of the peripheral nerves were affected, such as the median, sciatic, and external popliteal; microscopical examination showed an acute neuritis, similar to the inflammation of the cranial nerves.

Ophthalmoscopically, we usually find in the beginning of the neuritis more or less swelling of the papillæ, tortuosity of the vessels, and blurring of the edges of the disk. This is by no means always the case, for the ophthalmoscopical findings may be negative all through the disease. The retina is usually not affected. Haziness in the region of the macula and an increasing pigmentation of the pigment epithelium have been described. Microscopical examination does not explain the latter condition. Some authors consider these changes as due to a degeneration of the retina.

Both optic nerves are involved in the majority of instances. Two cases, however, are recorded in which only one of the nerves was diseased, but in one of these cases there was an overfilling of the vessels of the opposite eye.

After the disturbance of vision has reached its highest stage—this lasting, on an average, about two weeks—there is usually an improvement if the case is not fatal; and, rarely, a return of vision. Only three cases are on record in which the return of vision was complete. Usually complete blindness in both eyes results.

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[3] Rev. Neurol., Jan. 30, 1904.

Ophthalmologically, in about half the cases there is a resulting white atrophy of the optic nerves; neuroretinal changes have been observed in two instances, and in three the ophthalmological findings after the subsidence of the ocular symptoms were completely normal, but in none of these three cases were the symptoms severe.

Remissions of the symptoms have been noted in five instances. These occurred simultaneously in the eye and in the cord, and have been explained in various ways. Elschnig[4] believes that the pathological process in the optic nerve is in the nature of a primary interstitial neuritis; and that the inflamed interstitial tissue exerts a mechanical pressure upon the nerve-fibres and the vessels, thereby interfering with their function. Should the inflammation subside, function would be restored. Bielchowsky, however, thinks that the process is a primary parenchymatous neuritis; and that the remissions occur because the toxin causing the disease is diminished, and the axis cylinders regenerate, thus restoring function.

Of the 39 reported cases of myelitis associated with optic nerve changes, 20 have been with autopsy. It is striking that in nearly all the myelitic process involved almost the whole extent of the spinal cord. The process in all the cases was either acute or subacute, both in the spinal cord and in the optic nerves, and not chronic.

In most instances the nature of the inflammation is that of an acute disseminated myelitis. Transverse myelitis is of rare occurrence. Histologically, the myelitis is either in the nature of an acute diffuse parenchymatous inflammation or a combined parenchymatous and interstitial process. Bielchowsky,[5] in a thorough microscopical examination of four cases, found three distinct pathological processes. In his first case the inflammation was of the acute hemorrhagic type, affecting the whole extent of the spinal cord. Here the inflammation was as much interstitial as parenchymatous. In the second case, the myelitis was diffuse and of a primary parenchymatous nature, and affected principally the white matter. The nerve-fibres were everywhere destroyed, the myelin being partially or completely broken up, and the axis-cylinders were degenerated. The lumina of the blood-vessels were not overfilled, but the walls, especially the adventitia, were full of granular cells. The glia fibres seemed normal or perhaps a little swollen, and the neuroglia cells were increased in number. In the meshes of the glia tissue, however, there was an enormous number of granular or fat-cells, these being also very numerous around the blood-vessels. A slight proliferation of the endothelial cells of the intima was likewise present. In the third and fourth cases the inflammation seemed to be limited largely to the posterior and lateral tracts. Histologically, the process was similar to that in the second case. In the majority of instances of myelitis with optic neuritis, the myelitis is such as is reported in Bielchowsky's second case—notably so in the cases recorded within recent years, in which microscopical examinations have been made by modern methods. Some authors, however, as Elschnig, interpret the inflammation as being primarily interstitial in nature.

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[4] Arch. f. Augenheilkunde, vol. xxvi.

[5] Myelitis u. Sehnervenentzündung, Berlin, 1901; V. v. S. Karger.

The histological process in the optic nerve is always similar to that in the spinal cord. There is either an acute hemorrhagic inflammation or a primary diffuse parenchymatous neuritis. In the latter case, however, the inflammation does not present the diffuse areas seen in the spinal cord, but a continuous lesion. This is due to the merging of the adjacent softened areas.

In most instances the optic nerve is involved throughout its whole extent, from the papillæ to the chiasm and through the whole thickness of the nerve. This is not always so, however, for there may be in the periphery of the nerve a small band of normal fibres. The chiasm is involved in most cases, and the process may extend back of the chiasm into the optic tracts. Secondary degeneration may be traced partially into the optic tracts, but the primary optic centres—*viz.*, the external geniculate body, the pulvinar of the optic thalamus, and the anterior corpora quadrigemina—have never been found diseased. The fibres of Gratiolet and the cuneus have also never been found in a diseased condition.

A cross section of the diseased optic nerve will always show a widening of the interfascicular septa; an increase in the number of neuroglia cells; a large number of flattened granular cells in the meshes of the glia tissue and around and in the blood-vessels; and a destruction of the nerve-fibres. As a rule, the pial covering of the optic nerve is not altered. It has been observed to be diseased, however, in two instances; and here it was probably local in origin, for the accumulation of nuclei was opposite the point of greatest inflammation of the optic nerve. The endothelial cells covering the subarachnoid spaces were also increased in these two cases.

If the whole optic nerve is equally diseased, it is impossible to determine whether the process is of retrobulbar origin or not. In the majority of instances, however, the optic neuritis is retrobulbar in origin, for in an examination of the limits of the pathological process —as in the chiasm—recent changes may be found. The inflammation, however, need not implicate both the intraorbital and intracranial portions of the optic nerve; for in a number of cases the intraorbital part of the nerve was perfectly normal,—the inflammation beginning in the central part of the nerve, before its decussation, and involving the chiasm and the optic tracts to a limited extent.

Marked neuritic atrophy of the papilla and of the surrounding part of the retina has been described by Dalen.[6]

Examination of the other involved cranial nerves showed an intense acute neuritis. The inflammation was not secondary to a nuclear change nor to an inflammation of the pia, and there was no secondary degeneration.

In two instances—those of Elschnig and of Weil and Gallawardin[7] —there was a slight diffuse interstitial encephalitis. In no case was there basal meningitis. In the medulla oblongata, except for the usual ascending secondary degenerations, nothing abnormal was found.

Relation between the Myelitis and the Optic Neuritis.—Several theories have been advanced to explain the occurrence of optic neuritis with myelitis. Wharton Jones believed that the optic neuritis was caused by a circulatory disturbance, due to involvement of the cervical sympathetic. Allbutt is of the opinion that the optic neuritis is due to an ascending basal meningitis; while still other authors believe that an accompanying intracranial lesion causes the optic neuritis.

From the evidence of recent pathological findings, all these theories may be dismissed. In most instances the optic neuritis precedes the myelitis; so the hypothesis of an optic neuritis secondary to a cervical-sympathetic lesion is not tenable. Again, there is not the accompanying sympathetic phenomena; nor does a sympathetic lesion produce optic neuritis.

Allbutt's theory may be readily dismissed, for no basal meningitis has been found in any recorded case. So far no secondary intracranial lesion has been observed. In only two instances was there cerebral involvement, and in these it was in the nature of a diffuse encephalitis.

Taylor and Collier[8] have recently attempted to show that the optic neuritis is always due in these cases to a lesion of the cervical part of the spinal cord; and that in every one of the reported cases the pathological process involved the cervical cord. A careful analysis, however, does not seem to bear out this statement. In sixteen of the twenty autopsies the lesion did extend to the cervical cord; but in the majority of these the symptoms seem to have indicated that the pathological process had begun in the dorsal cord and extended upwards. Clinically there was no analogy between the appearance of the optic neuritis and the myelitis. In fact, as before stated, the optic neuritis generally precedes the myelitis. In support of this theory it may be said that in several reported cases the optic neuritis was most marked on the side corresponding with the greater involvement of the spinal cord, the lesion in these cases being principally in the cervical cord. Taylor and Collier also believed that the inflammation must be a partial one; that is, not involving the whole thickness of the cord.

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[6] Arch. f. Ophthalm., vol. 48.

[7] Rev. Neurol., Oct. 31, 1903.

[8] Brain, 1901.

In view of the fact that the histological process is alike in the spinal cord and in the optic nerve, we must come to the conclusion that the processes are identical and due to the same cause—probably a toxin that exerts its influence equally on the spinal cord and on the optic nerve. Why this toxin should pick out the optic nerve is, however, a question. It may be explained on the theory of *locus minoris resistentiæ*. The optic nerve has the same embryological origin as has the spinal cord, and it may be that the toxin that acts upon the spinal cord has a selective action upon the optic nerve.

Prognosis.—As a rule, the prognosis in these cases depends upon the progress of the myelitis, for improvement in the spinal-cord symptoms always indicates an improvement in those of the optic nerve.

Treatment.—Treatment is usually unavailing. When a history of syphilis is obtained, which is in about one-third of the cases, a combined antisyphilitic treatment is indicated; but, as a rule, this is of no benefit. The bed-sores should be carefully looked after, and the contracted limbs kept in good condition. Catheterization is commonly indicated.

SYRINGOMYELIA.

As a rule, the symptom complex of syringomyelia is so typical that the diagnosis does not offer much difficulty. The disease generally begins early in life and is chronic and progressive. The symptoms may be divided into three forms: first, the atrophy of the muscles of the upper limbs; second, the disturbances of sensation, and, third, vasomotor and trophic phenomena.

The atrophy usually begins in the small muscles of the hand and may involve all the muscles of one or both upper limbs, but is not uniform. Fibrillary twitching may be present and electrical reactions of degeneration may be obtained.

Sensation for touch is generally preserved, but pain and temperature sensations are lost. This dissociation of sensation may be present in one or both upper limbs and part of the trunk, depending upon the part of the spinal cord involved, but it is usually more marked on one side.

Vasomotor and trophic phenomena, as cyanosis of the hand, œdema, arthropathies, and deformities of bone, are common.

The lower limbs may be spastic, due to secondary degeneration of the pyramidal fibres, or may be paralyzed, due to direct involvement of the lumbar cord, but this is exceptional. If the disease involves the medulla oblongata and pons, we may have symptoms of cranial nerve palsy, as atrophy of the tongue, disturbances of respiration, laryngeal palsy, deafness, facial paralysis, and symptoms of involvement of the different ocular nerves.

Pathology.—The cause of this symptom complex is a cavity or a gliomatous tumor formation in the substance of the spinal cord, which generally involves its centre, the posterior columns, or the posterior horns. It hardly ever involves the anterior part of the cord, and may be unilateral throughout its whole extent. The cavity is usually largest at the cervical cord and ends in the lower thoracic region, thus explaining the preponderance of symptoms in the upper limbs. It has been thought that the cavity does not extend higher than the nucleus of the seventh nerve in the pons, but Spiller has observed an instance in which the cavity extended as high as one cerebral peduncle. It is important to remember this fact, especially when we consider the cause of the different ocular nerve palsies. The cavity is filled during life with cerebrospinal fluid. A congenital defect in the formation of the spinal cord is probably the basis of the disease.

Ocular Symptoms.—Eye symptoms are uncommon. We shall consider in order involvement of the optic nerves, the ocular muscles, and the pupils. Optic neuritis has been recorded by Saxer,[9] Bullard and Thomas,[10] and by Thorington and myself.[11] In all of these cases a complicating internal hydrocephalus was probably at fault, as was shown by the necropsy findings in the first two. It is well to remember that internal hydrocephalus is not an infrequent complication of syringomyelia, and that many of the cranial-nerve palsies which may be present in syringomyelia are due to the pressure of the cerebrospinal fluid. This was the case in the above-mentioned instances, in which both external recti and the facial nerve were diseased. Hoffmann suggests that a tumor situated in the medulla oblongata, which may be the starting point of a syringomyelic cavity, may cause optic neuritis. This undoubtedly may occur and it is probable that in some of the cases of secondary optic atrophy this may be the explanation.

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[9] Beiträg. z. path. Anat. u. z. allgem. Path., vol. xx. No. 11.

[10] American Journal of Medical Science, 1899.

[11] Ibid., Nov. 1905.

Primary optic atrophy occurs less often in syringomyelia than in any other spinal-cord disease. Maixner (quoted by Schlesinger), in thirty-two cases, found optic atrophy in three. In one case under my observation gray degeneration of the optic disk occurred in one eye, the other fundus being fairly normal. This is unusual, for, when disturbances do occur, they usually involve both optic nerves equally. It must be remembered, however, that tabes dorsalis rarely complicates syringomyelia, and when primary optic atrophy is present this disease should be excluded.

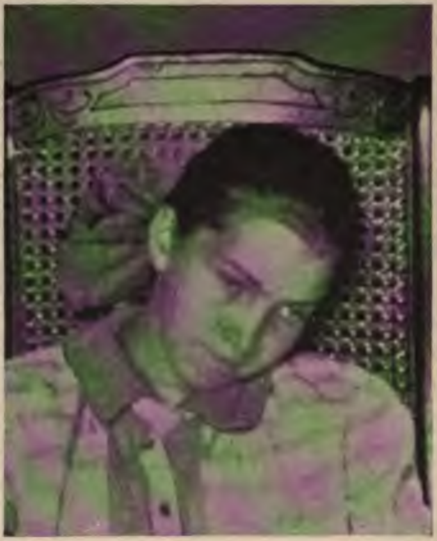

FIG. 2.—Case of syringomyelia with optic neuritis and bilateral external rectus paralysis; also showing position of head. (Reported with Dr. James Thorington.)

The ophthalmoscopic findings are usually negative. The disks may be pale, but always in their whole extent, differing from the temporal paling so often seen in multiple sclerosis. The fundi in two of my cases were gray in color. Vision, as a rule, is not interfered with, but if disturbances occur, both eyes are equally affected. Neither scotoma nor disturbances of light and color sense have been recorded in this disease.

A concentric restriction of the visual fields, especially for colors, and particularly for green, sometimes occurs. Schlesinger[12] collected from the literature one hundred and thirty cases, in which this symptom was present in thirty-eight. The examinations were perimetric. Of these, twenty-four had no symptoms of hysteria. The contraction of the visual fields was not severe, and consisted in a peripheral defect, especially for green. He concludes that in most cases of syringomyelia the visual fields are normal, that the contraction of the fields may be due to a concomitant hysteria, and that in a small number of cases of syringomyelia not complicated with hysteria, alterations in the visual fields may occur.

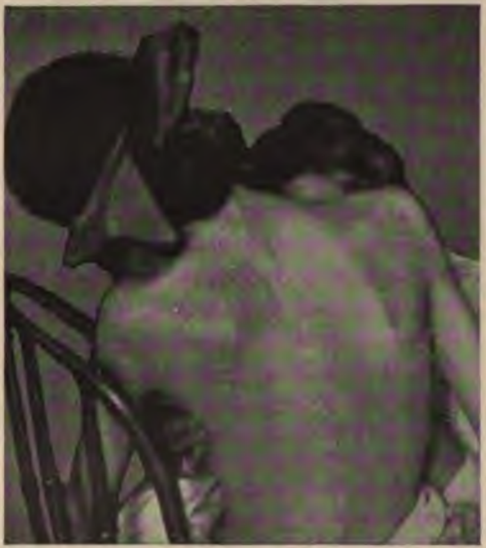

FIG. 8.—Case of syringomyelia with optic neuritis, showing lateral curvature. (Reported with Dr. James Thorington.)

Other authors, however, do not believe that in an uncomplicated case with negative ophthalmoscopic findings visual contractions can occur, and ascribe these symptoms to hysteria. In five cases under my observation, in which a perimetric examination was made, no contraction of the visual fields was noticed. Schlesinger explains the contraction by an intracranial complication, probably a hydrocephalus. If this is so, we might expect that these symptoms would vary on different examinations, but this is not mentioned. In the eight cases recorded by Dejerine and Tuilant,[13] who were the first to call attention to the restriction of the visual fields, an examination a year later still denoted the same condition.

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[12] Die Syringomyelie, Leipzig u. Wien, 1902.

Paralysis of Ocular Muscles.—Schlesinger, in a study of three hundred cases from the literature, found paralysis of the ocular muscles in thirty-three, approximately in 11 per cent. of the cases. It occurs less often than in multiple sclerosis and tabes dorsalis. The paralysis generally comes on early in the disease and may be transient, but later in the disease may become permanent. These transient paralyses resemble those of syphilis and tabes.

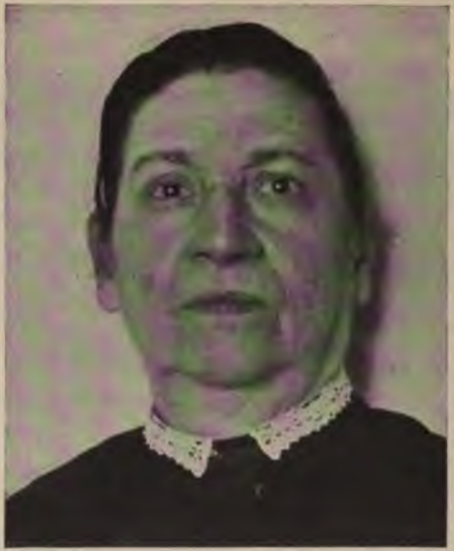

FIG. 4.—Case of syringomyelia with partial ptosis of right upper lid.

The abducens nerve is more often involved than the other cranial nerves, and more often the involvement is unilateral. The oculomotor nerve is usually only partially involved, as shown by a ptosis. This is present in the patient whose photograph is given. The ptosis may be slight and may vary from day to day, as in the patient in the photograph, in whom the ptosis varied considerably on successive examinations. This variability in the amount of paralysis, as shown by the abducens nerve, seems to be characteristic of syringomyelia.

The ptosis which is due to the third nerve involvement must be differentiated from the ptosis due to sympathetic paralysis. It is well known that in sympathetic paralysis all of the symptoms need not be present, and we may have in both conditions only a ptosis. An application of cocaine will, in sympathetic paralysis, not dilate the pupil.

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[13] Semaine Med., 1891, No. 6.

If the syringomyelic cavity extends high up into the pons, we may have complete chronic external ophthalmoplegia. Retraction of the ocular muscles appears late in the disease.

A direct involvement of the ocular nuclei or of their intramedullary fibres by the cavity will explain the ocular palsies, providing there are in these cases some other symptoms of bulbar paralysis. If they are not present, an internal hydrocephalus is probably the cause. The transient ocular palsies and variability of the ptosis may be explained by the difference in pressure of the fluid in the syringomyelic cavity.

According to Schlesinger, nystagmus and nystagmoid movements of the eyeball in syringomyelia occur in about 15 per cent. of the cases. Of three hundred and thirty cases, fourteen had true nystagmus and thirty-seven nystagmoid movements. Schlesinger states that the nystagmus comes on early in the disease, but it may be a late manifestation. The movements may be horizontal, vertical, diagonal, or rotatory. Disturbance of vision in nystagmus never occurs, but in a number of instances the fixing object was said to be fluctuating.

The nystagmoid movements are far more common than those of true nystagmus. They are obtained only on lateral deviation.

The cause of the nystagmus and the nystagmoid movements in this disease are not known, but various theories are given. Some authors believe that they are an accidental complication of the disease, but it may be argued against this hypothesis that the nystagmus comes on as the disease appears. Schlesinger believes that it is due to the pressure of hydrocephalus, while other authors believe that it is congenital.

In the different diseases in which nystagmus is a symptom, such as Friedreich's disease and in syringomyelia, a congenital anomaly in the formation of the brain and spinal cord is thought to be the cause. Multiple sclerosis also is believed by some to be produced in the same way. In these diseases the symptoms do not appear until some contributory cause starts the pathological process. Nystagmus appears at the same time. It is a striking fact that in syringomyelia, in the cases in which nystagmus occurs, there are no disturbances of vision, accommodation, or in the pupillary reflex, this arguing against an acquired lesion. It is probable, therefore, that the cause of the nystagmus in these diseases is some congenital maldevelopment, and that this symptom does not appear until the symptoms of involvement of the rest of the brain and spinal cord present themselves.

Pupillary Symptoms.—The pupils in syringomyelia are usually not affected, but myosis and differences in the size of the pupils sometimes occur. In two hundred cases studied by Schlesinger pupillary differences were found in twenty-four. The pupillary light reflex is not often absent, and yet a number of such cases have been recorded. Dejerine and Mirallié record an instance where the light reflex was absent on one side only. It may be assumed that when the pupillary light reflex is disturbed or absent, we may have an accompanying tabes or general paresis.

Paralysis of the sympathetic may be caused by a cavity in the lower portion of the cervical cord. Sympathetic symptoms are generally unilateral, but may be bilateral. Such symptoms are narrowing of the palpebral fissure, myosis, enophthalmos, and disturbances of the secretions. As a rule, we do not have all of these symptoms in sympathetic paralysis, and we may have only myosis with ptosis without involvement of the secretory functions. These symptoms are caused by the involvement of the sympathetic fibres which ascend in the spinal cord from the ciliospinal centre in the first dorsal segment to the medulla oblongata.

Involvement of the Fifth and Seventh Nerves.—The descending sensory portion of the trigeminus nerve is sometimes involved in syringomyelia. This is first manifested by paræsthetic phenomena, later by pains, and finally by anæsthesia. A disturbance in the secretion of tears and of saliva has been noted in a few instances. The secretion of tears is sometimes increased and appears on laughing or on irritation of the nasal mucous membranes on the side opposite the anæsthesia. The corneal and lachrymal reflexes are lost. Trophic disturbances are uncommon. This may be due to an incomplete paralysis of the trigeminus, but even in total paralysis trophic disturbances may not occur. This is probably due to the nature of the lesion, for it has been shown that an irritative condition, either in the pons or in the Gasserian ganglion, may produce a keratitis. According to Gowers,[14] a greater irritation is necessary to produce a corneal ulcer if the lesion involves the fifth-nerve fibres within the pons or the sensory root than if the lesion is at or in front of the Gasserian ganglion.

The facial nerve is sometimes involved in syringomyelia. The paralysis may be transitory. All or only part of the nerve may be diseased. If the upper portion is much involved, it causes a lagophthalmos.

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[14] Diseases of the Nervous System, vol. ii. 217.

THE RELATION OF OCULAR SYMPTOMS TO SPINAL-CORD DISEASES.

In the following pages will be considered the various ocular disturbances occurring in conjunction with certain spinal-cord diseases, such as primary lateral sclerosis, amyotrophic lateral sclerosis, posterolateral sclerosis, and the subacute combined system degenerations which sometimes complicate pernicious anæmia. Primary ascending unilateral paralysis will also be considered in this connection. As a rule, ocular changes are rare, but optic-nerve atrophy has been recorded in all of these diseases, as well as pupillary phenomena.

To understand the relation of the optic-nerve atrophy to the various spinal-cord diseases, we must first clearly understand the nature of these spinal cord affections. The belief is gradually gaining ground that such diseases as primary lateral sclerosis and amyotrophic lateral sclerosis are due to a defect of vital endurance, or, as Gowers has recently termed it, to an "abiotrophy." Both of these diseases come on, as a rule, early in life. The optic nerve atrophy appears simultaneously with the spinal-cord symptoms, and both manifestations are uniformly progressive. We are led to believe, therefore, that we have in the optic-nerve atrophy the same primary cause—a lack of vital endurance. It is difficult to explain, however, why the optic nerve or certain tracts of the spinal cord should be picked out for degeneration.

Lack of vital endurance may be manifested late in life, as, for instance, in such diseases as paralysis agitans. It is possible that this explains the unilateral slowly progressive ascending paralysis described by Charles K. Mills.

Such degenerations as are due to pernicious anæmia are undoubtedly toxic in origin. The poison which is generated by the disease circulates in the blood and acts upon the nervous structures. It may be due to a lack of resisting power that the spinal cord and the optic nerves are affected.

Tabes dorsalis is also a disease which is produced by a toxin, and it is most probable that posterolateral sclerosis, which comes on late in life, is due to a similar cause—a syphilitic toxin—and that the cause of the optic-nerve atrophy in the two affections can be similarly explained.

It is noticeable that in all diseases just mentioned, the optic-nerve atrophy is progressive, and that return of function does not occur. Ophthalmoscopically, we find a gray degeneration of the disk. The retina, as a rule, is not affected. There is usually a peripheral restriction of the fields, and there may be a central scotoma.

The pupillary phenomena are attributed to the disturbance of the cervical sympathetic. This will be later fully discussed.

PRIMARY DEGENERATION OF THE PYRAMIDAL TRACTS (PRIMARY LATERAL SCLEROSIS).

In this affection both pyramidal tracts are diseased, the nerve cells in the anterior horns of the spinal cord being normal. Both lower limbs are usually involved, and we have the well-known picture of lateral sclerosis. The limbs are weak and very spastic. All the tendon reflexes are increased, and walking is usually impossible because of the weakness and the contractures. There is no atrophy of the muscles. As the disease progresses, the upper limbs become involved similarly to the lower.

As a rule, there are no eye symptoms; but, rarely, optic atrophy similar in character to that seen in tabes is observed. Gowers mentions a case of spastic paraplegia in a boy of twenty, who, besides the spinal-cord symptoms, had a gradually developing primary optic atrophy. The acuity of vision was reduced to one-tenth. The fields presented a peripheral restriction. There was a well-marked central scotoma for red and green, which was irregular in outline, but extended to an average of about ten degrees from the fixing-point, and, in one eye, to twenty degrees.

The degeneration in the pyramidal tracts and in the optic nerves was simultaneous and progressive in character, and argued for the abiotic nature of the disease. Again, the eye symptoms were similar to those seen in an ordinary atrophy of spinal origin. Optic neuritis is said to have occurred in the course of lateral sclerosis, but an examination of the recorded cases fails to show clearly such an occurrence.

AMYOTROPHIC LATERAL SCLEROSIS.

In this form of primary disease of the motor system there is, in addition to the clinical picture of lateral sclerosis just described, an atrophy of the muscles of the affected limbs. This is caused by the involvement of the nerve cells of the anterior horns in the spinal cord. The disease is progressive, and finally involves the different nuclei in the medulla oblongata and pons, giving, besides the spastic symptoms, bulbar phenomena, such as difficulty in speech and deglutition, dribbling of saliva, atrophy of the tongue, limbs, and face, and fibrillary twitching of the parts involved.

If the disease is limited to the spinal cord, eye symptoms are rare. When the bulb becomes involved, there may be any of the ocular phenomena common in bulbar palsy, which will be described in another chapter.

Irregularity of the pupils, with partial or total loss of reaction to light, and optic atrophy have been described. The pupillary phenomena are explained by the sympathetic involvement; while the optic-nerve atrophy is similar to that seen in other primary degenerations of the pyramidal tracts. Spiller mentions a weakness of one external rectus muscle in one instance.

PROGRESSIVE ASCENDING UNILATERAL PARALYSIS.

This disease, which has only recently been described by Mills, is a form of primary degeneration of one pyramidal tract, ascending in character. A lower or an upper limb is first affected; then, in the course of some time (in the original case, eighteen months), the other limb is involved, giving the picture of a hemiplegia. A weakness of the lower distribution of the face is also present. The affected limbs are weak and spastic, and the reflexes are increased.

In a case subsequently described by Spiller, in which the eye examination was made by Dr. Hansell, the report was as follows:

Right Eye.—Media, clear; fundus, normal. A myopia of 3 D. exists.

Left Eye.—Media, clear; arteries, small; veins, normal. The edges of the disk are distinct. The nerve-head is white, the atrophy probably being spinal in origin. There is no indication of previous neutritis.

It is interesting to observe that in this case the atrophy of the optic nerve corresponded with the primary degeneration of the pyramidal tract of the same side, both being on the left; whereas the other optic nerve was normal.

POSTEROLATERAL SCLEROSIS.

In this combined system disease there is a degeneration of the posterior and lateral tracts of the spinal cord. Besides the increase of the reflexes, the spastic condition, and the weakness of the limbs that are common in lateral sclerosis, there is a spastic-ataxic gait, incoördination of the limbs, disturbance of sensation for touch and pain, especially on the sole of the foot, in front of the tibia, and on the chest as is common in tabes, and a disturbance in the functions of the bladder and the rectum.

Ocular Symptoms.—Rigidity of the pupil to light is unusual, but Gowers [15] has observed it in two or three cases. In one instance, accommodation was lost, the light reaction being normal. It may be said that pupillary changes occur in the cases in which syphilitic infection is present. Optic-nerve atrophy is rare, although it has been observed in a few cases. Usually there is no involvement of the extraocular muscles, although nystagmus has been recorded.

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[15] Diseases of the Nervous System, vol. i. 506.

I have had under my care a man about thirty years of age, who, besides the symptoms of posterolateral sclerosis, showed, one year after the beginning of these symptoms, the following eye phenomena:

The pupils were three millimetres in width. The direct and consensual reactions were normal. There was incipient optic-nerve atrophy. Both optic nerves were conspicuously pallid and slightly excavated. No gross fundus changes were observed in the retina or the choroid. On lateral deviation to the right or the left, decided nystagmoid movements were observed. The examination was made by Dr. E. A. Shumway.

It is possible that true nystagmus does not occur in this disease, but we have what may be called nystagmoid movements, if we choose to make such a distinction. In nystagmus the movements may be in any direction and to either side of the fixing point, whereas in nystagmoid movements the oscillations of the eyeball are always horizontal and are obtained only on lateral deviation.

SPINAL-CORD SYMPTOMS IN PERNICIOUS ANÆMIA.

It is well known that we may have changes in the spinal cord due to any chronic anæmic condition, and this has been especially well recognized in pernicious anæmia. Spinal symptoms may appear even before the blood-count indicates the disease, but as a rule they are manifested after the disease has well advanced, or they may come on simultaneously.

Pathologically, there is a diffuse degeneration of the posterior and lateral columns, but the degeneration is not limited to these tracts, and extends beyond these areas to the anterior part of the cord. The process is uniform and involves symmetrical areas.

Besides the usual symptoms of pernicious anæmia, as weakness, debility, and reduction in the amount of hæmoglobin and in the red and white blood-cells, the disease usually begins by a feeling of numbness, or a pins-and-needles sensation in the legs, followed by weakness and spasticity. The reflexes are increased; in a short time the upper limbs become similarly involved, and we have, besides, a considerable ataxia of all the limbs and a disturbance of the bladder and rectal functions. Later there may be complete loss of sensation, a flaccid condition may succeed the spasticity, and all the reflexes may be lost.

Ocular Symptoms.—Eye symptoms are rare. Towards the end of the affection the pupils have been found unequal, and in one instance there was a slight bilateral ptosis and a marked myosis due to sympathetic involvement. In a few instances reflex iridoplegia has been reported. Nystagmus has also been observed. Optic-nerve atrophy is extremely rare, only a few cases being on record.

I have under my observation an interesting case of this kind. The patient, a man of 42, without any previous ailment, suddenly had a gastric hemorrhage. The following day he began to lose sight in both eyes, and in a week he was completely blind, in which condition he has remained. He did not experience any weakness in his limbs until one day after sight was completely lost, from which time he gradually developed the typical spinal symptoms of the affection. The blood-count showed the usual changes of pernicious anæmia.

Ophthalmoscopic examination, by Dr. de Schweinitz, made four days after the blindness became complete showed a double optic neuritis. The pupils were dilated and were irresponsive. There was shrinking of the retinal arteries and overfilling of the veins, and there was some œdema of the nerve-head and surrounding area.

It is hard to explain the evident low-grade optic neuritis which was present in this case, as shown by the examination, and the relation it bore to the sudden loss of vision. A gastric hemorrhage may bring on an atrophy of the optic nerve and cause blindness in a few hours, but it will not produce optic neuritis. Most probably there was a low-grade optic neuritis present before the hemorrhage came on, but this did not seriously affect the vision, and the sudden loss of blood brought on the rapid amaurosis. Optic neuritis in the course of pernicious anæmia may occur, but it usually gives a picture of an albuminuric retinitis, and the low-grade optic neuritis may have been a preliminary stage of the affection. Ophthalmoscopical examination at the present, ten months after the onset of the disease, shows a typical post-neuritic atrophy. There are also nystagmoid movements on lateral deviation.

INJURIES OF THE SPINAL CORD.

Injuries to the substance of the spinal cord are by no means rare. Fracture and dislocation of the vertebræ are the most common cause. Dislocation of the vertebræ is most frequent in the cervical region, between the atlas and axis and between the fifth and sixth cervical vertebræ. The spinal cord may be injured although the vertebræ are not at all harmed. There may be at the moment of injury a sudden torsion of the cord, which may produce a hemorrhage, or a softening, or both.

Usually the hemorrhage, or hæmatomyelia, is in the central part of the spinal cord. Direct pressure causes softening of the affected parts, and there may be also hemorrhages and areas of softening either above or below the point of greatest pressure. Gunshot injuries or stab wounds are not unusual. The spinal cord may be directly injured or there may be a resulting hemorrhage and softening from injury to the vertebræ.

Symptoms.—The symptoms produced will depend entirely upon the part of the spinal cord involved. If there is a fracture or dislocation, there is usually a deformity of that part, accompanied by rigidity of the back and neck, and pain on movement. All of the limbs are paralyzed if the injury is above the cervical enlargement; if below this part, only the lower limbs are paretic. The reflexes are at first lost, and later become increased. The rectal and bladder functions are disturbed, and sensation is lost, depending upon the area of the cord destroyed. The symptoms of traumatic myelitis are identical, except that we have no dislocation or fracture of the vertebræ. In central hæmatomyelia the same picture is presented, and we may have, besides, the syringomyelic disturbance of sensation, and the pain may be excessive.

We shall here also consider such other diseases which produce symptoms of compression or disturbance of the function of the spinal cord, because the ocular phenomena are identical. Such diseases are tumors of the vertebræ and of the spinal cord, aneurism of the aorta, caries, carcinoma of the vertebræ and the meninges, and pachymeningitis cervicalis hypertrophica.

Tumors of the vertebræ are far more common than tumors of the meninges or of the substance of the cord. Whether the tumor is extramedullary or intramedullary, the symptoms are usually identical, and depend upon the part of the cord involved. The pain in these cases is sharper and more persistent than in any other spinal disease, and its location depends upon the part of the body supplied by the involved spinal segment or spinal root.

An eroding aneurism of the descending aorta, caries of the vertebræ, carcinoma of the vertebræ and of the meninges, and pachymeningitis will give symptoms of pressure upon the spinal cord, depending upon the part involved, and, as these symptoms in the main resemble each other, we will not consider each separately. The differential diagnosis can be readily made in most instances by the concomitant symptoms.

Ocular Symptoms.—There is no subject on which we have less accurate information than the ocular symptoms accompanying injuries of the spinal cord. Most of the literature consists of a repetition of statements made by a few authors, and these are by no means definite. Considerable confusions exists, partly because the ocular symptoms following an injury of the spinal cord are confused with the ocular manifestations due to a hysterical or neurasthenic condition following trauma of the spine, the so-called traumatic neurosis.

In the latter condition we may have dimness of vision, pain in the head and eyes, reversion of the color-fields, and a concentric diminution of the fields of vision, and any of the hysterical phenomena accompanying the disease. This subject will be fully discussed in another chapter.

The ocular phenomena, when they follow a true injury to the spinal cord, may be divided into three classes: first, changes in the optic nerve; second, sympathetic phenomena; and, third, pupillary changes. These will be discussed in order.

Symptoms of Involvement of the Optic-Nerve.—Clifford Allbutt,[16] in 1870, first called attention to the changes in the fundus oculi in injuries to the spinal cord. This author stated that these phenomena, which consisted in a slight haziness of the disk and a distention of the retinal veins, did not occur except in chronic cases, and only in the course of a few weeks or months, and that the higher the lesion the more likely are these symptoms to occur. These findings are somewhat at variance with later reports. Thorburn recorded three cases of injury to the cervical cord with ophthalmoscopical changes, but in his cases the symptoms appeared in the acute and not in the chronic cases. Taylor and Collier [17] record two cases of fracture and dislocation. In one case there was a definite optic neuritis in one eye, apparently caused by a piece of bone pressing upon the lower cervical cord. When this was removed the neuritis rapidly disappeared.

The early report of Allbutt is by no means definite, as we are not told how often and when the eye examinations were made. Again, our knowledge at that early period, 1870, was by no means as definite as it is at present, and we doubt somewhat the statement that changes in the fundus occur only in chronic cases.

It is probable that at times changes in the disk occur, but these are mild and temporary, and, therefore, frequent and early ophthalmoscopical examinations are necessary to detect the condition. Similar changes have been described in rare instances in tumors of the cervical cord, as in a case of Taylor and Collier, of an extramedullary myxoma comprising the third cervical segment. Here there was a double optic neuritis. In caries of the cervical vertebræ we may have slight changes in the fundus, but in the cases where there is a definite optic neuritis an intracranial tubercle probably is the cause.

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[16] The Lancet, Jan. 15, 1870.

[17] Brain, 1901.

It is curious that no ocular disturbances occur in any case in which the lesion is below the second dorsal segment. A few cases are on record where lesions below this area produced changes in the fundus, but a closer examination always showed involvement of the cervical cord. We are led, therefore, to believe that in some cases of injury to the spinal cord we may have a low-grade optic neuritis, and that this only occurs if the cervical cord is involved. It is difficult to explain this. We have seen that optic neuritis occurs in conjunction with myelitis, and this has been explained by a similar pathological lesion in the optic nerve and in the spinal cord due to the same cause. But here we have a traumatic myelitis of the cervical cord, as a result of which there are at times mild and sometimes definite changes in the optic nerve. This is further shown in the instances in which unilateral compression of the cervical cord produced unilateral changes in the fundus.

Wharton Jones explained this relation by a sympathetic involvement; but this is not correct, because in lesion of the cervical sympathetic we do not have optic neuritis. At present no satisfactory explanation can be offered; further study—especially more accurate and frequent ophthalmoscopic examinations in every case of injury of the spinal cord and experimental lesions on lower animals—is necessary to solve this interesting problem.

Symptoms of Sympathetic Involvement.—The cervical sympathetic is quite frequently involved in traumatic lesions of the spinal cord. Our knowledge of the anatomy and physiology of the sympathetic system is by no means exact. Through experimental lesions upon lower animals by Budge and others and by clinical experience, we know that in the lower part of the cervical and the upper part of the dorsal cord there is a so-called ciliospinal centre. This area has been more accurately localized in the eighth cervical, and especially in the first dorsal segment. The anterior roots of the first dorsal segment seem to contain the oculopupillary fibres. This has never been better demonstrated than by Oppenheim, who had the opportunity of stimulating with an electric current the exposed anterior roots of the first dorsal segment in a case of gunshot injury to the spine. Oculopupillary symptoms were present on stimulating these roots, while stimulation of the roots below had no such result. The eighth cervical root was not exposed.

The classical symptoms of irritation of the cervical sympathetic are enlargement of the pupil, widening of the palpebral angle, a slight exophthalmos, delayed descent of the upper lid on looking downwards, paleness of the face, and increase of the sweat secretion. Paralysis of the cervical sympathetic produces a small pupil, narrowing of the palpebral angle, a slight enophthalmus, a warmth or coldness of the face, and hyperidrosis or anidrosis. Intraocular tension is somewhat diminished.

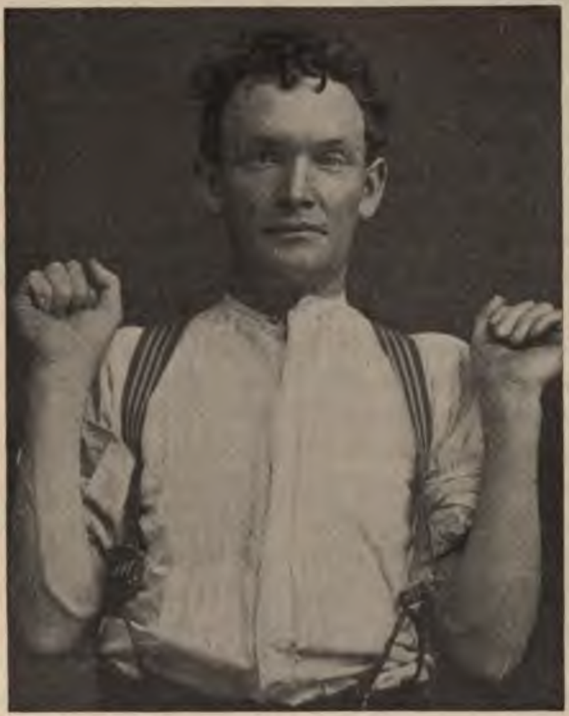

FIG. 5.—Case of hemorrhage or traumatic myelitis in the left eighth cervical and first dorsal segments, causing Brown-Séquard symptoms, with sympathetic paralysis of the left side, narrowing of pupil, narrowing of palpebral fissure, retraction of eyeball, and paralysis of the left hand and forearm. The photograph shows the patient's inability to firmly close the left hand. (Case of Dr. W. G. Spiller, Polyclinic Hospital.)

We, however, rarely see all of these symptoms in a lesion of the sympathetic. This is partially explained by the different centres in the spinal cord for the oculopupillary and the vasomotor, trophic, and secretory functions. C. Bernard demonstrated in lower animals that by cutting the first dorsal roots oculopupillary phenomena only were produced, while cutting the sympathetic fibres coming from the second to the fourth thoracic vertebræ, produced vasomotor symptoms. It is generally thought that the vasomotor fibres are transmitted from the third to the sixth dorsal roots, inclusive.

Oppenheim's case is proof of this, for there were no vasomotor symptoms on irritating the first dorsal roots. Kraus[18] is of the opinion that there are only oculopupillary symptoms if the spinal cord is principally affected, but if vasomotor symptoms predominate, the rami communicantes of the sympathetic are more involved.

The oculopupillary fibres leave the ciliospinal centre by means of the rami communicantes of the first dorsal root and join the inferior cervical sympathetic ganglion. Here these fibres are joined by the vasomotor fibres from the second to the sixth thoracic segments, inclusive. By means of the cerebral sympathetic, these fibres enter the orbit and innervate the dilator pupillæ, the non-striped part of the levator palpebræ superioris, the orbital muscle of Müller, and a small bundle of non-stripped muscle which lies behind the globe of the eye and bridges across the sphenomaxillary fissure at the back of the orbit.

In the majority of instances, the only sympathetic phenomena observed in traumatic lesions of the cervical cord are a myosis, and sometimes a narrowing of the palpebral fissure. These symptoms are not only produced if the lesion is in the eighth cervical and first dorsal segments, but as well if the lesion is higher. We do not know the course of the fibres of the cervical sympathetic in the upper cervical cord, bulb and the brain, but it is probable, as Kocher[19] thought, that these fibres run together in the eighth cervical segment and then diverge in their course cerebralwards. Therefore, a lesion above the eighth cervical segment would produce only partial symptoms, as myosis and ptosis, because only a number of the sympathetic fibres would be involved. A lesion limited to one side of the cord will produce unilateral sympathetic symptoms.

Gunshot injuries of the cervical sympathetic sometimes occur. In most of these instances the symptoms produced are those of paralysis of the sympathetic accompanied by vasomotor phenomena. Tumors of the substance of the cervical cord itself, and extramedullary tumors pressing upon the cord or upon the rami communicantes may produce sympathetic phenomena. Growths of the glands of the neck, intrathoracic tumors, especially those of the upper posterior mediastinum, and aneurisms of the descending aorta, subclavian, common carotid and vertebral arteries have also been known to produce symptoms of sympathetic involvement, but these instances are rare.

Pupillary Changes.—In a number of instances of injury to the upper cervical cord the pupils have been found irresponsive to light, while the reaction to accommodation and convergence was normal. The study of the cause of reflex iridoplegia has very recently taken on an additional interest, because of the experiments of Bach and Meyer.[20] While their conclusions have been based upon experiments on lower animals, and as yet clinical evidence has not substantiated their investigations, still, considering our meagre knowledge of the subject, such an important contribution should be carefully considered.

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[18] Zeitsch. f. Klin. Med., 1905, vol. xviii.

[19] Die Läsionen d. Rückenm., 1896. Jena.

These authors believe that in the upper part of the spinal cord and the lowest part of the floor of the fourth ventricle, near the median line, there is an inhibition centre for the light reflex, and an inhibitory centre for pupillodilation. Irritation of this part always produced pupillary rigidity and myosis. A right-sided irritation produced reflex iridoplegia upon the left side. Bach also explained the myosis which always accompanied the reflex iridoplegia by a more powerful action of the sphincter over the weaker dilator pupillæ, assuming that the irritation of the inhibitory light reflex centre had aborted all other irritation in the pupil, placing it in a state of absolute rest.

In a series of cases of tabes and general paresis Reichardt [21] attempts to prove that the reflex iridoplegia occurring in these diseases, and also in cases of fracture and dislocation of the upper cervical vertebræ, and in tumors of that region, are dependent upon the lesion in the spinal cord. He examined cases with and without failure of the light reflexes, and in every instance where there was pupillary rigidity he found a degeneration of the endogenous fibres, especially of the second and third cervical segments, in the area between the posterior commissure and the ventral part of the column of Goll, the so-called Bechterew's intermediate zone.

Reichardt assumes that there are four pupillary centres in the cervical cord: First, Budge's centre, the so-called ciliospinal centre in the lower cervical cord; second, a pupillodilator centre in the upper part of the cervical cord; third, an inhibition centre for the light reflex; and fourth, a centre for dilation of the pupil by irritation of the skin covering the cervical region in the fourth and fifth cervical segments. Reichardt evidently accepts Bach and Meyer's experiments proving an inhibition centre for the light reflex as conclusive.

This author assumes that the centres two and three, which are pupillo-dilators and pupillo-contractors, respond to all kinds of irritation, both psychical and sensory, thus causing a constant change in the pupil. In destruction of the endogenous fibres of the posterior cord there is brought about an abnormal condition and a consequent failure of the light reflex. An interruption of the fibres between centres one and three will cause the same result, presuming that the course of these fibres is in the posterior columns.

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[20] Arch. f. Ophthalm., vols. lv. and lvi.

[21] Arch. f. Psych. u. Nervenkr., vol. xxxix. No. 1.

Reichardt does not believe that a traumatic lesion of the upper cervical cord produces immediate pupil rigidity, because the endogenous fibres would not have time to degenerate, but there probably would be loss of the light reflex just before death. In chronic cases, as in a bulbar syringomyelia, there would be destruction of these endogenous fibres and a consequent pupillary rigidity. The endogenous character of the lesion must be shown by an absence of degeneration of the posterior roots in the upper dorsal and cervical cords. This author also calls attention to the absence of pupillary rigidity in traumatic lesions of the upper dorsal and lower cervical cords, for the lesion to produce this sign must be proximal to the fourth cervical segment. A number of cases of fracture and dislocation and tumors of the cervical cord, with pupillary rigidity, are quoted to prove the assertion of the author.

These statements cannot be definitely accepted without further proof. It would be well if in all cases of tabes dorsalis and injury to the upper part of the spinal cord, there were a thorough microscopical examination made of the uppermost part of the cervical cord, for it is more than probable that there is some relation between this region and the condition of the pupil and the optic nerve.

In a case of dislocation of the third cervical vertebræ with a resulting myelitis of the fifth cervical segment, the pupils were noted to be unequal, and a day before death there was no reaction of the pupils to light. Examination of the second, third and fourth cervical segments failed to show any degeneration in any part of the sections.

ACUTE ASCENDING PARALYSIS (LANDRY'S PARALYSIS).

Considerable confusion still exists as to the true nature of this disorder, first described by Landry in 1859. According to Landry the symptoms begin with a diminution of power in the lower limbs, which spreads rapidly and successively involves the muscles of the buttocks, loins, abdomen, chest, arms, and of the neck. The limbs are flaccid, the reflexes are lost and there may be some paræsthetic phenomena, but no other sensory disturbances. The bladder and rectum are never involved, and the mind remains clear to the end. The course is rapid and may be fatal in a few days, either through respiratory or cardiac paralysis.

This symptom-complex has been modified to a considerable degree, and while we still recognize the rapidly ascending form of the paralysis, other symptoms may be present. It can be assumed that the symptoms just described are to be found in the early stages of the disorder, and, if it be not fatal, we may have added anæsthesia of the limbs, abdomen and chest, pain on pressure over the nerve trunks, atrophy of the muscles, reactions of degeneration, and, sometimes, paresis of the functions of the bladder and rectum. If the medulla oblongata becomes involved, we have added symptoms of bulbar palsy, as difficulty of speech and inability to swallow.

The etiology of the affection is still obscure. It has been known to follow cold, influenza, various infectious diseases, and other similar causes. The pathological findings in some cases have been entirely negative, but in most of the recent autopsies alterations in the nerve-cells of the anterior horns of the spinal cord, and an engorgement of the vessels of the cord and the leptomeninges have been found. In some instances diffuse inflammation of the spinal cord has been described. In most cases a parenchymatous degeneration of the peripheral nerves is present, and often these were the only pathological findings, causing some authors to believe in the peripheral origin of the symptom-complex.

It is probable that the disorder is due to an acute intoxication, and the toxin may be caused by various agents, and this toxin acts upon the entire central nervous system. This toxic theory is supported by the uniform finding of an enlarged spleen and liver, and the febrile symptoms which usually usher in and accompany the paralysis. Bacteriologically a number of bacilli have been found, but none characteristic. Buzzard was able to isolate a bacillus from the blood of a patient with Landry's palsy which, when cultivated and injected into rabbits, caused the symptoms of an acute ascending paralysis.

Ocular Symptoms.—Eye symptoms in Landry's paralysis are uncommon, because the affection is usually fatal before the nuclei of the eye muscles are involved. Abducens paralysis has been noted. Three cases are on record in which ptosis was present. In the case of Hoffmann[22] the patient had difficulty in articulation and in swallowing, and the tongue movements were weak. Both facial nerves were paretic, and there was a right-sided ptosis. Bailey and Ewing[23] recorded ptosis in another instance. Goebel,[24] in a brief report, mentions involvement of all the eye muscles, besides difficulty in swallowing, chewing, and talking. In these three instances the autopsy showed a diffuse myelitic process of the medulla oblongata. In Goebel's case, which lasted for seventeen days, myelitic degeneration by the method of Marchi was found in the area between the oculomotor nucleus and the pyramidal crossing.

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[22] Arch. f. Psych., vol. xv.

[23] New York Medical Journal, 1896.

[24] Neurolog. Centralbl., 1898, No. 7.

Pupillary symptoms are equally rare. Paresis of accommodation has been reported by Bernhardt in one instance, and unequal pupils in another. Slow reaction to light may also be present.

In our present knowledge of Landry's symptom-complex it is difficult to give a clear explanation of these ocular disturbances, but it is probable that there is a nuclear or fascicular involvement of the affected cranial nerves.

DISEASES OF THE NERVES.

In this connection we shall consider only the diseases of the brachial plexus, because in this type of paralysis the rami communicantes of the first dorsal roots may be involved and cause symptoms of sympathetic paralysis. Vasomotor phenomena are not usually present, because the probable course of these fibres is through the second to the sixth thoracic roots.

Paralysis of the brachial plexus may be total or partial, unilateral or bilateral. If total, the arm hangs limply by the side, no movements being possible; the muscles are atrophic, electrical reactions of degeneration are obtained, and trophic phenomena are present. Partial brachial paralysis may be either of the upper plexus type, the so-called Duchenne-Erb form, in which the fifth and sixth cervical roots or the fibres in the plexus coming from these roots are involved, or the Klumpke, or lower plexus type, in which the eighth cervical and first dorsal roots are diseased.

In the upper plexus form the deltoid, triceps, brachialis anticus, the supinator longus and brevis, and the infraspinatus muscles are diseased. It is impossible to adduct the arm, and the forearm is extended and pronated. Sensation, as a rule, is not disturbed. The muscles are atrophic, and there may be reactions of degeneration. In the Klumpke paralysis the small muscles of the hand and a number of the muscles of the forearm, especially the flexors, are paralyzed. Sensory disturbances are common in the hand and forearm, especially in the ulnar distribution.

These different types of brachial plexus paralyses are mostly traumatic in origin, and may be due to blows, gunshot or stab wounds, fracture of the head of the humerus, dislocation of the shoulder, and tumors. The so-called obstetrical and narcosis paralyses are included in the upper-arm type. The disease may also occur idiopathically, the cause probably being toxic.

Ocular Symptoms.—Oculopupillary symptoms, consisting in a narrowing of the pupil and of the palpebral fissure, can occur only if the first dorsal roots are involved, either in the intervertebral foramen, or before their separation from the rami communicantes. Therefore, in the lower arm type of paralysis, in which the eighth cervical and first dorsal roots are diseased, we always have oculopupillary symptoms. It is possible, however, to have this type of paralysis without sympathetic involvement, if the fibres coming from these roots, in the brachial plexus, and not the roots themselves, are diseased. It is difficult, however, to make such a clinical differential diagnosis because the symptoms are identical, but we can always assume that, if oculopupillary symptoms are present, the first dorsal root is diseased.

In involvement of the fifth and sixth cervical roots in the intervertebral foramen, oculopupillary symptoms may occur. Heubner [25] records three such cases. In the first, there was an osteosarcoma of the seventh and eighth cervical vertebræ; in the second, caries of the seventh and first dorsal vertebræ; and, in his third case, there was a tumor involving the same area. This can be explained only by the accidental involvement of the rami communicantes of the first dorsal root, or by involvement of the sympathetic fibres in the spinal cord. In disease of the fifth and sixth cervical roots outside of the intervertebral foramen, oculopupillary symptoms were noted in a case by Oppenheim, in which a tumor of the supraclavicular region pressed upon the fifth and sixth cervical roots, and in another similar instance of Levy Dorn's. Here there must have been an involvement of the oculopupillary fibres in the cervical sympathetic.

In the Duchenne-Erb type of paralysis, due to a birth palsy, or the paralysis occurring in the course of etherization, the traction upon the arms may cause an abnormal stretching and tearing of the rami communicantes of the first dorsal root, thus causing sympathetic paralysis without the first dorsal root itself being diseased.

If all of the roots of the brachial plexus are diseased, we may have oculopupillary symptoms. As a result of gunshot or stab wounds we may have forms of paralysis which do not conform with any of the known types, with sympathetic symptoms. In these cases the first dorsal roots are either involved, or the oculopupillary fibres in the cervical sympathetic are injured.

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[25] Charité Annalen., 1895.

Eulenburg and Guttman do not believe that sympathetic phenomena accompany every brachial plexus palsy, but are of the opinion that in these cases of traumatic neuritis the inflammation spreads to the spinal cord through the involved roots, causing a circumscribed secondary myelitis, and that this myelitis occurring in the lowest part of the cervical and first dorsal segments causes oculopupillary symptoms. While this may be true in some instances, it is not the rule, for experimental investigations have demonstrated that cutting the first dorsal roots will cause immediate oculopupillary phenomena.

MULTIPLE NEURITIS.

Multiple neuritis is produced by a variety of causes, chronic alcoholism being the most common. Toxic disturbances, such as are produced by the various infectious diseases and the different metallic poisons, the chronic diseases such as tuberculosis and malaria, beriberi, leprosy and senility, may produce polyneuritis. The disease may come on without any apparent cause or follow a cold, it probably being in these instances infectious in origin.

The symptoms of multiple neuritis may develop suddenly or subacutely. The patient first complains of a feeling of numbness in his feet or hands, or of sharp pains, these gradually increasing in severity. A gradual weakness in the lower or the upper limbs then appears, but sometimes the loss of power may come on very suddenly. There may be accompanying febrile symptoms, and the patient may be delirious. The muscles are flaccid and the tendon and skin reflexes are abolished. There is tenderness and pain on pressure over the nerve trunks and the pains may become very acute. Sensation is usually affected, there being first hyperæsthesia followed by anæsthesia. The paralysis is symmetrical and involves in the lower limbs principally the peroneal and anterior tibial nerves, causing bilateral foot drop, and in the upper limbs the distribution of the radial, median and ulnar nerves, causing wrist drop. The paralysis is degenerative in character and in a short time electrical reactions of degeneration are usually obtained. The bladder and rectum are hardly ever involved, this being an important diagnostic feature. Vasomotor and trophic phenomena are sometimes present.

At times ataxia of the upper and lower limbs is present and the patient is unable to walk because of this. This form of multiple neuritis is sometimes called peripheral neuro- or pseudo-tabes and it is at times impossible to distinguish this disease from tabes dorsalis. We have not, however, in the former the chronic onset, the Argyll-Robertson pupil, the girdle sense and the involvement of the bladder and rectum, this being, perhaps, sufficient to make a different diagnosis.

The cranial nerves are usually not affected in the course of multiple neuritis, but instances of multiple neuritis limited to the cranial nerves have been noted. The ocular nerves are more often involved than the other cranial nerves, especially the sixth nerve. The facial nerves are sometimes diseased, especially in conjunction with the sixth nerve; more rarely the trigeminus and acoustic nerves are involved. The vagus is more commonly affected, especially in severe cases, causing an increase of the pulse rate, and sometimes death. The involvement of the ocular nerves will later be fully discussed. The symptoms above described are largely common to all forms of multiple neuritis; the special forms will be taken up separately and the ocular symptoms accompanying them will be dwelt upon.

Alcoholic Multiple Neuritis.—The symptoms above described are essentially those of alcoholic multiple neuritis. We sometimes have in addition a peculiar psychic involvement which consists of loss and confusion of memory for recent events, and in addition illusions and hallucinations. This is sometimes called Korsakoff's psychosis.

Pathologically, we have an inflammatory or parenchymatous degeneration of the different peripheral nerves, especially of the muscular and sensory branches of the skin. It is not uncommon, however, to find in this disease an alteration in the cells of the anterior horn and of the medulla oblongata and pons. These changes in the nerve-cells consist of a displacement of the nucleus to the periphery, and a central chromatolysis. Diffuse and degenerative inflammations with hemorrhages have also been noted, especially in the region of the gray matter of the third ventricle and of the aqueduct of Sylvius. This was first described by Wernicke,[26] who termed the disease acute hemorrhagic polioencephalitis superior and inferior, depending whether the upper or the lower cranial nuclei were involved. A degeneration of the anterior and posterior roots and of the posterior and anterior horns has also been described. These changes, however, are not to be confused with those of multiple neuritis, in which we have only alterations in the peripheral nerves.

Alterations in the muscles are not uncommon. These may consist only of a narrowing of the muscle fibres, or they may be more severe, causing a loss of striation of the fibres and increase of the nuclei. Thomsen [27] described in one case similar alterations in an eye muscle.

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[26] Lehrbuch der Gehirnkrankh., vol. ii. 1881.

[27] Arch. f. Psych., 1890.

Ocular Symptoms.—Ocular symptoms are not very rare. We shall take up first the alterations in the optic nerve, then the pupillary phenomena, and lastly the involvement of the motor cranial nerves of the eye.

Disturbance of sight is uncommon, but when it does occur it is generally in the nature of a central scotoma, especially for colors. Blindness has been reported in a number of instances, but in none is there an uncomplicated history. In the clearest case, that of Render,[28] the ophthalmoscopic examination was not given, and a number of the other cranial nerves were involved. In the other reported cases albumin was always found in the urine.

Optic Nerve.—The optic nerve is very seldom involved in alcoholic multiple neuritis. A pathological whitish paling of the temporal side of the disk has been described, especially by Uhthoff,[29] who examined one thousand alcoholics and found such changes in 139. He found, by histological examination, a retrobulbar neuritis in six of these instances. It cannot be assumed, however, that the pathological paling of the temporal side of the disk depends upon the retrobulbar neuritis, for either condition can occur without the other being present. Optic neuritis is an exceedingly rare occurrence. I have been able to find in the literature only seven recorded cases. The neuritis may be well marked in both optic nerves, or may be limited to one. It is usually very mild in character, but may be very severe. The neuritis comes on generally late in the disease and in most instances disappears as the disease improves. In all of the recorded cases, except in one, a part or all of the third, sixth and seventh nerves were also involved.

I have had under my observation such a case in the wards of the Philadelphia Hospital. This man, forty years of age, has been drinking steadily for the past eighteen years, mostly whiskey, sometimes as much as twenty-five drinks a day. He has had delirium tremens once, six years ago. He smoked somewhat, but denies venereal history. Four months ago he first began to have pain in both eyes and complained of photophobia and of a discharge from both eyes. Dimness of vision also became apparent in both eyes about the same time. The pain was relieved somewhat by the wearing of blue glasses, but sight has gradually diminished. He never had diplopia. Examination of the eyes by Dr. E. A. Shumway was as follows:

R. V. 5/30. L. V. 5/35. Subacute conjunctivitis in both eyes.

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[28] The American Lancet, 1890.

[29] Arch. f. Ophthalm., vols. xxxii. and xxxiii. 1887.

The pupils are equal and respond promptly. The fields are normal. No color scotoma. Green is recognized badly. Ophthalmoscopic examination. Right eye: Media clear, low grade optic neuritis. Left eye: Similar low grade optic neuritis, with fine haze in the vitreous, posterior part.

The patient had dimnution of power in all of his limbs; the reflexes, while not lost, were much diminished; there was pain on pressure over the nerve trunks and spontaneous shooting pains in the limbs. He has had previous attacks of loss of power in his lower limbs and has complained of pain for a long period. The urine examination showed a very slight trace of albumin. The neuritic symptoms have improved, but the optic neuritis still persists, and sight is no better.

In these cases we must exclude the occurrence of a large amount of albumin in the urine, for a nephritic condition might readily cause the optic neuritis. In only one of the recorded cases, that of Thomsen,[30] was there a pathological examination. Here the examination of the optic nerve was not mentioned, although the author states that the examination of the brain and the spinal cord was entirely negative and that the disease was limited to the peripheral nerves.

It is difficult to explain why the toxic process caused by the alcohol should have a selective action upon certain peripheral nerves, and still more difficult to explain why it should involve the optic nerve, which is not really a nerve at all. As a rule, intoxications which affect the peripheral nerves will not affect the optic nerve. From the insufficient number of cases on record and the absence of pathological and experimental investigations, we cannot assume that the optic neuritis is caused by the toxin of alcohol. It is probable that the optic neuritis which is present may be due to the chronic meningitis which is present in some alcoholics.

Pupils.—The pupils are usually not affected in alcoholic multiple neuritis, but a difference in size is not unusual. I have seen this in a number of instances. Uhthoff in an examination of one thousand alcoholics found a difference in the size of the pupils in sixty of his cases. In twenty-five there was, besides, a slow reaction of the pupil to light, and in ten instances there was a reflex pupillary rigidity, the reaction to accommodation being nearly always preserved. Oppenheim records reflex iridoplegia in both pupils, and in one eye accommodation was also lost. Eperon records a similar instance without, however, loss of accommodation. Moeli is of the opinion that pupillary rigidity is not permanent in alcoholics, it being transient in character. I have also observed this in one instance.

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[30] Neurolog. Centralbl., vol. vi. No. 1.

Ocular Nerves.—The eye muscles are not often involved. Unilateral external rectus palsy is the most common form of ocular paralysis, but both abducens nerves may be paralyzed. The seventh nerve is at times also involved, generally with the sixth nerve. In two patients recently under my observation, external rectus palsy was present in each, while in one the left seventh nerve was also slightly paretic. In one patient, hearing was for a time diminished. The fifth nerve is sometimes also diseased. Dr. Spiller very kindly furnished me with the records of a case of alcoholic polyneuritis in which the exit points of the fifth nerve were tender to pressure and the patient complained of a feeling of numbness in his face. This disappeared in a short time.

One or both oculomotor nerves may be involved. More often the outer musculature of the oculomotor nerve alone is diseased, causing ptosis, while the inner musculature remains free, iritic motion and accommodation not being disturbed. Edinger explained this peculiar involvement by the theory that the external muscles are paralyzed because more strain is thrown upon them by the poison. This, however, is not a satisfactory explanation.

Uhthoff, in his one thousand alcoholics, found paralysis of the ocular muscles in twenty-two cases: in four there was transient diplopia, and in two bilateral abducens paralysis. The ocular palsies usually appear in the height of the disease, in about the third or fourth week, and are usually transient. In my cases the abducens palsy came on in the second week and lasted for about two weeks, and then disappeared. In one four weeks afterwards the bilateral abducens palsy reappeared with a weakness of the left seventh nerve. Besides, the woman had difficulty in swallowing and she had the indistinct, nasal speech of bulbar palsy. The pupils were unequal and wider than normal and reacted to light and in accommodation. The neuritic symptoms were still severe. In the course of a few weeks the bulbar symptoms disappeared.

Pathology.—While the number of recorded cases of alcoholic multiple neuritis with microscopic examination is large, those with involvement of the ocular nerves are by no means common. Marina[11] recorded a case of a man of sixty-one with involvement of the left oculomotor and the right abducens and facial nerves, with the symptoms of multiple neuritis. A careful microscopical examination of the peripheral nerves and of the brain and spinal cord gave entirely negative results. It is well known, however, that this can occur in polyneuritis of toxic origin.

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[11] Ueber multip. Augenlähmung, Leipzig u. Wien, 1896.

A number of cases are recorded in which the microscopical examination showed a peripheral degenerative neuritis of the ocular nerves, without an involvement of their nuclei. Such, among others, are the cases of Dammront and Mayer, in which there was bilateral external ophthalmoplegia, besides an involvement of the seventh, ninth and twelfth nerves; of Thomsen, in which there was bilateral abducens palsy and ptosis, besides a nystagmus, and a mild left optic neuritis. In Thomsen's case the only branch of the oculomotor nerve diseased was the twig to the levator palpebræ muscle.

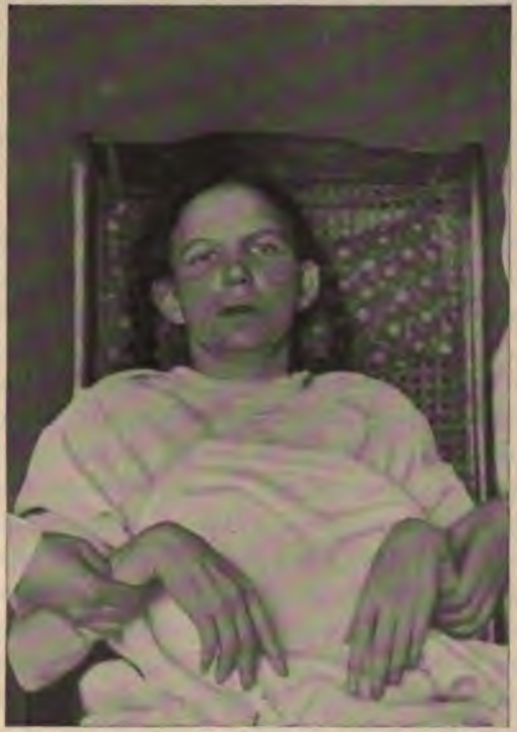

FIG. 6.—Case of alcoholic multiple neuritis with external rectus palsy and bilateral wrist-drop. The external rectus palsy is not shown in the photograph.

In a later report, Thomsen,[32] in a case of alcoholic polyneuritis with ocular symptoms, found the peripheral ocular nerves and their roots and nuclei intact, but found hemorrhages in the gray matter of the third ventricle, and came to the conclusion that the ocular palsies occurring in alcoholic multiple neuritis were due to a cerebral lesion, the polioencephalitis hemorrhagica superior of Wernicke. This observation has since been confirmed by others, as Boedeker and Rennert. Cases are on record, however, in which, besides this encephalitis, the cranial nerves themselves were the seat of a degenerative neuritis (Thomsen and Jacobæus). Again, Gudden[33] reported a case of polyneuritis in which the encephalopathic process involved the region of the third ventricle and the aqueduct of Sylvius without producing any ocular symptoms.

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[32] Arch. f. Psych., vol. xxi. Case III.

We may, therefore, have in alcoholic multiple neuritis: first, no pathological changes either in peripheral ocular nerves or in their central connections; second, alterations only in the peripheral ocular nerves; third, changes only in the region of the gray matter of the third ventricle and the aqueduct of Sylvius; fourth, a combination of the degenerative lesions of the peripheral ocular nerves with the encephalitic process of Wernicke.

Clinically, it is almost impossible to differentiate between these pathological processes. A rapid onset with a bilateral ophthalmoplegia, in which both the outer and the inner musculature are involved, with a possible, accompanying optic neuritis, may point to an acute polioencephalitis.

Nystagmus has been observed repeatedly in alcoholic polyneuritis, especially on lateral deviation. Such cases have been recorded by Oppenheim and others. Uhthoff, in his one thousand alcoholics, found in thirteen, nystagmoid movements on lateral excursion, and in two definite nystagmus. Thomsen in a similar examination of a number of alcoholics found nystagmus in five. I have observed similar nystagmoid movements in a number of my cases, but always on lateral deviation. External rectus palsy had been present. It has seemed to me that these were nystagmoid movements, due to the weakness of the abducens nerves and not true nystagmus. In none of the cases of polyneuritis was there nystagmus on direct fixation.

Lead Multiple Neuritis.—The effects of lead on the central nervous system are well known. Workers in lead, typesetters and others, who come in contact with this metal often suffer from this disease. The symptoms of lead neuritis are generally preceded by lead colic. The poison seems to have a peculiar affinity for the posterior interosseous, causing paralysis of the extensors of the hand and fingers, while the supinator longus and triceps muscles are usually spared. The nerves of the lower extremity are not, as a rule, involved, but if they are, the peroneal nerve is usually diseased, the tibialis anticus nearly always escaping. Sensation is not often disturbed. Ataxia hardly ever occurs. The muscles are atrophic and reactions of degeneration are soon obtained. A blue line on the gums is an aid to the diagnosis of lead palsy.

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[33] Arch. f. Psych., 1896.

In some instances lead poisoning causes intracranial symptoms; this is termed lead encephalopathy. Here we may also have paralysis of the cranial nerves and of the vocal cords and the laryngeal muscles. All of the ocular nerves, the third, fourth and sixth, may be involved, either alone or in combination. Either optic neuritis or atrophy may occur. Delirium, coma, convulsions, epileptic seizures, hemorrhages and transient hemiplegia have been observed in the course of lead encephalopathy. This subject is discussed in another chapter.

Pathology.—Pathologically, the predilection of the lead poisons for the peripheral nerves has been well established. The neuritis is degenerative in character. Alterations in the cells of the anterior horn, as described by Oppenheim, Spiller and others, may occur, and the anterior roots have also been found diseased. Goldflam described hemorrhages in the anterior and posterior horns. The muscles are the seat of degeneration.

In the literature there are many reported cases of involvement of the cranial nerves in the course of lead multiple neuritis, but in view of the scarcity of microscopical examination and our insufficient knowledge of the pathology of lead palsy, there is doubt regarding the existence of a pure, peripheral multiple neuritis with involvement of the cranial nerves.

Partial paralysis in the distribution of the vago-accessory and paralysis of the trigeminus, facial and the hypoglossus nerves have been reported, but always with involvement of one or more of the other cranial nerves. A mononeuritis of a cranial nerve is not on record. A critical analysis always shows, besides, symptoms of cerebral involvement. It is striking also that when facial paralysis does occur it is confined only to the lower distribution, this arguing for a central lesion.

A number of cases are noted in which the multiple neuritis was said to be confined only to the cranial nerves. Such are the cases of Pal,[34] Mannaberg, and Chvostek.[35]

In Pal's case a typesetter of twenty-one had for two years attacks of eclampsia, during which she had right-sided abducens and facial paralysis. After a severe attack of lead colic, disturbances of vision appeared, followed by optic neuritis and right-sided abducens and facial palsy, with electrical reactions of degeneration. Shortly total amaurosis with right-sided ptosis became apparent. There was also ataxia of the upper limbs with bilateral, peroneal palsy of the lower extremities. At the necropsy the gray matter, especially of the occipital lobes, the internal capsule and the medulla oblongata were harder than normal. No mention is made of a microscopic examination of these parts. The microscopical examination of the optic and the third, sixth and seventh cranial, the radial and peroneal nerves showed partial degeneration. In the spinal cord there was a degeneration of the posterior roots and of some of the tracts.

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[34] Ueber multip. Neuritis, Wien, 1891.

[35] Wiener. klin. Woch., 1898, No. 52.

In the two cases reported by Mannaberg there was headache, dizziness, bilateral optic neuritis, right-sided facial palsy and right-sided neuralgia, limited to the distribution of the fifth nerve in one case, while in the other there was bilateral neuroretinitis and right-sided facial and abducens palsy.

In Chvostek's case there was paralysis of both oculomotor, the left abducens and the right facial nerves and bilateral optic neuritis. There were also severe cerebral symptoms. At the necropsy the brain substance was extremely œdematous, the convolutions were flattened and the oculomotor and abducens nerves were found degenerated. This degeneration was probably due to the pressure caused by the œdema.

In every one of these cases there were some symptoms of involvement of the peripheral nerves, besides symptoms of cerebral involvement. It is probable that a careful microscopical examination of the brain and cord would have shown the alterations found in lead encephalopathy. It is improbable, considering the nature of the poison and its widespread effects upon the cerebrospinal system, that the cranial nerves should be affected without involvement of the brain substance. We can conclude, therefore, that multiple neuritis, limited to the cranial nerves, does not occur, and that if in the course of a lead polyneuritis, symptoms of involvement of the cranial nerves appear, the brain itself is diseased.

Optic neuritis alone has been observed repeatedly in the course of chronic lead-poisoning. Goldscheider[36] has called attention to the interesting fact that optic neuritis may occur alone in conditions which lead to polyneuritis, as, for instance, in the infectious diseases and in the different metallic poisons. It is probable, however, that in these cases the optic neuritis is not a precursor of a multiple neuritis, but that it is a manifestation of a cerebral disease. As has been previously mentioned, the optic nerves are not, as a rule, affected by the same toxic processes that are prone to involve the peripheral nerves.

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[36] Charité Annalen, vol. xvii.

Changes in the size of the pupils, with loss of the light reflex, have been observed, but these are probably also encephalic symptoms.

Paralysis of the eye muscles without involvement of the other cranial nerves is not very common in lead-polyneuritis. In 1898 Elschnig[37] collected sixteen cases from the literature. In these the eye muscles were either unilaterally or bilaterally involved. In four instances the oculomotor nerve was totally paralyzed. The trigeminus and the abducens nerves were also involved in several instances. There was, however, in these cases no clear history, and syphilis could not be excluded in several instances. Here also we may assume that these paralyses were only a part of the general lead encephalopathy.

Arsenical Neuritis.—Arsenic is a frequent cause of polyneuritis, but I have been unable to find any reference to involvement of any of the ocular muscles. Optic neuritis has been reported by Higier, but it is probable that there was a cerebral involvement in his case.

Polyneuritis due to other Metallic Poisons.—Mercury, copper, phosphorus, and carbon disulphide have also been known to produce multiple neuritis, but these instances are rare.

The form of polyneuritis due to carbon disulphide is extremely uncommon and little is known of its pathology, as observations are lacking. Workers in vulcanized rubber are especially prone to this disease. Mental excitation or depression with hysterical manifestations precede the neuritis phenomena, which are, as a rule, of the ataxic form and resemble greatly the alcoholic type of neuritis. Hysterical symptoms are so common that some authors, as Marie, insist that the hysterical manifestations are among the most prominent symptoms.

Ocular phenomena are common and consist, as a rule, of alterations in the visual fields, especially for colors. Amblyopia is a fairly constant symptom. Scotoma either large or small is found in some cases. Pupillary rigidity and even nystagmus have been recorded. In this country within the year 1905 Jump and Cruice were the first to report two such cases, one of which I have had the privilege of studying. These cases showed marked hysterical symptoms. The eye examination by Dr. Langdon of the first case showed the optic disks to be slightly congested with hyperæmia of the retinal vessels. The visual fields were much contracted.

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[37] Wiener. med. Wochen., 1898, Nos. 27–29.

It is probable that we have here not a pure multiple neuritis but a toxic process which so influences the central nervous system as to produce the various mental, ocular and neuritic symptoms. It must be acknowledged that most of the various manifestations shown in this disease are hysterical in nature; but why should a previously healthy individual who is poisoned by carbon disulphide be hysterical only so long as the influence of the poison lasts?

The influence of toxins, whether generated within or without the body, upon the brain and spinal cord is becoming better recognized. It is more than probable that their influence is not selective but general, and that we have alterations not only in the peripheral nerves, but also in the central nervous system. Such is the case, for instance, in lead or alcoholic poisons and in uræmia.

Carbon Monoxide Neuritis.—Illuminating gas, or carbon monoxide, may produce symptoms which resemble multiple neuritis. Involvement of the different cranial nerves is not uncommon. Cases are recorded in which the third, fourth, fifth, sixth, and seventh nerves were paralyzed either wholly or in part. The paralysis persists only for a short time and disappears as the general symptoms improve. Blindness has also been recorded. Sibelius reported an interesting case of a boy of twenty, who, after exposure to carbon monoxide for five minutes, became unconscious. He regained consciousness the following day when it was found that the boy was blind. The pupils were large and reactionless and the left seventh nerve was paretic. Subsequently weakness of the extremities developed with dementia and epileptic seizures. The ocular symptoms persisted until death, which occurred three months after. The microscopical examination showed a diffuse encephalitis and a disease of the blood-vessel walls, but no thrombosis. The amaurosis was explained by a bilateral area of softening in the occipital lobe.

This case illustrates well that toxic processes do not produce local alteration in the peripheral nerves, but that they have a general action on the whole central nervous system.

Sulphonal and Trional Neuritis.—Since the introduction of coal-tar derivatives, poisoning by these drugs is becoming known. This is especially so with sulphonal. Erbslöh recorded an instance of a man of forty who had taken two grammes of sulphonal daily for five days and developed symptoms of polyneuritis. Besides, the right pupil was larger than the left, the left abducens nerve was paralyzed and he had hallucinations of sight. At the necropsy the peripheral nerves were found degenerated. Dillingham also records ptosis, which lasted about ten days.

Diphtheritic Paralysis.—Approximately about one-quarter of the total number of cases of diphtheria are followed by paralysis. It is more liable to follow a severe attack, although paralysis has been known to follow a simple sore throat or diphtheritic inflammation elsewhere in the body. The older the person the greater is the tendency to paralysis. (See Chapter V, p. 231.)

As a rule, the paralysis does not appear until the diphtheria has entirely disappeared, in the third or fourth week and sometimes later, although in rare instances it may occur in the height of the disease. The symptoms of diphtheritic paralysis will not here be discussed, and we shall only consider the ocular phenomena.

Ocular Symptoms.—Loss of accommodation with impairment of vision for near objects, due to paralysis of the ciliary muscles, is the most frequent form of paralysis, next to that of the palate. It generally follows involvement of the latter or appears at the same time in about the third or fourth week. It is the most common of the ocular palsies. The ciliary involvement may be so slight that its presence may not be detected, especially in children. The patient may not be able to thread a needle or read, but may follow the finger.

The interference with vision will depend upon the normal refracting power of the eyeball. In a normal eye near vision will be interfered with; in hypermetropia this will be even more marked, while in a myopic eye there may be very little or no appreciable disturbance of vision for near objects, for accommodation is unnecessary.

The pupils, as a rule, are of normal size and the light reflex is never lost, although it may be sluggish. Slight contraction of the pupils in accommodation may be present, even though accommodation is entirely lost, and the former is rarely absent. Loss of accommodation generally comes on in two or three days and lasts two or three weeks and then disappears. It involves both eyes, but may affect one before the other. Ophthalmoscopic examination is always negative.

Paralysis of the ocular muscles is uncommon. Statistics upon this point vary, some authors giving as much as twenty-five per cent. of ocular palsies following diphtheria. The external and internal recti muscles are most frequently involved. This involvement may be unilateral, but is commonly bilateral and follows the loss of accommodation. The palsy may be so slight as not to be noticed, or may be fluctuating in character. The paralysis is usually of short duration.

The oculomotor nerve, either wholly or in part, is rarely involved. Ptosis is mentioned only in a few instances. Uhthoff records in a boy of ten double external ophthalmoplegia, without involvement of the pupils, with a mild ptosis. This lasted a month. Mendel and Gowers mention two cases in which almost all of the muscles supplied by the oculomotor nerves were affected. In Gowers' case there was also concentric contraction of the visual fields and, according to him, this is not so infrequent in diphtheritic palsy, for it generally escapes attention, due to the presence of the cycloplegia.

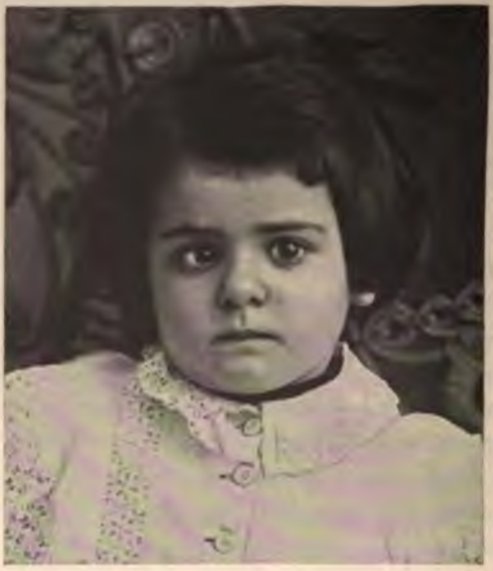

FIG. 7.—Case of diphtheria, with bilateral external rectus palsy, developing two weeks after the subsidence of diphtheritic symptoms, and lasting only two weeks. (Case from the nervous dispensary of the Philadelphia Polyclinic.)

The prognosis, so far as ocular palsies are concerned, is good. Only in very rare instances does the paralysis persist. Gowers mentions two cases in which there was a slow, progressive degeneration of certain cranial nerves occurring some time after the ordinary palsies appear. In one case, besides weakness of the lips, tongue and pharynx, there was almost complete internal and external ophthalmoplegia and primary atrophy of the optic nerves. In his second case sight began to fail six months after the diphtheria and was almost lost at the end of the year. Two and a half years after the primary disease, there was gray atrophy of the optic disks, left internal ophthalmoplegia, and a weakness of some of the movements of both eyeballs, especially upwards. These changes were stationary.

Harris[38] has recently recorded three cases in which bulbar symptoms followed a simple sore throat. In all of these cases the progress of the paralysis was typical of post-diphtheritic palsy. Besides the bulbar symptoms, the patient could not close the eyes properly, or early in the morning could not open the eyes. The symptoms were suggestive of myasthenia gravis, but Harris believes that they were diphtheritic in origin.

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[38] Brain, vol. xxvi.

Pathology.—The pathology of diphtheritic paralysis is still very much in doubt, because of the varied findings. As a rule, in the majority of cases the changes are confined to a degeneration of the peripheral nerves, especially in their distal parts. Usually, the neuritis is degenerative, but may be interstitial in character. These changes, however, are by no means found in every case. Gaucher records a case in which the brain, spinal cord, and peripheral nerves were entirely normal, while Hochhaus notes an instance in which the alterations were limited entirely to the muscles. Changes in the cells of the anterior horns of the spinal cord, hemorrhages in the gray substances of the spinal cord, increase of the neuroglia cells, and degeneration of the anterior and posterior roots have also been found.

Changes in the bulbar nuclei with a degenerative neuritis of the cranial nerves have been found. Such cases have been recorded by Mendel, P. Meyer, and others. In Meyer's case there was a disappearance of the axis cylinders, changes in the myelin and increase of the nuclei of the endoneurium of the third nerve. In one case there were minute hemorrhages in the walls of the sixth nerve.

Thus, from the pathology we cannot come to a definite conclusion whether the cause of diphtheritic palsy is central, peripheral, or both. We have, however, a definite disturbance of function which is caused by a poison circulating in the blood. It is only rational to believe that in such a toxic process we may expect alterations in all parts of the anatomy. This is borne out by the pathological findings. It is well known that certain poisons have a peculiar predilection for certain parts of the nervous system; in this case it is shown by the loss of accommodation. Wilbrand and Saenger believe that the cycloplegia is due to a nuclear or central lesion. It is difficult to believe, however, that the diphtheritic poison could involve only that portion of the oculomotor nucleus which is concerned in innervating the ciliary muscles. It is more probable that we have here a peculiar peripheral process which involves principally the nerve-fibres, and secondarily the muscles.

Multiple Neuritis due to Infectious Diseases.—Such infectious diseases as erysipelas, typhoid fever, pneumonia, measles, scarlet fever, gonorrhœa, influenza and rheumatism may cause multiple neuritis, but these instances are necessarily rare. Eye symptoms are exceedingly uncommon. Fuchs, however, records mild grade optic neuritis with paling on the temporal side of the disc in a typhoid neuritis. Microscopic examination showed a retrobulbar neuritis. It is probable that the infection did not directly cause the optic neuritis.

Influenza, or la grippe, may cause various nervous symptoms, among which is a mononeuritis of the different cranial nerves. Such are the cases recorded by Pflüger of optic neuritis, by Wilbrand and Saenger, Uhthoff and others, of paralysis of the different ocular muscles. Wilbrand and Saenger [39] reviewed the literature and collected the following cases of ocular paralysis occurring after influenza. In thirteen cases the third nerve was involved; in fourteen the abducens, and in nine the fourth. In twelve cases there was accommodation paralysis, and in one the iris was paralyzed. Ptosis occurred eight times. In five it was bilateral, in three unilateral. It is probable that in influenza we have a toxic process which acts directly upon the different cranial nerves, causing a temporary paralysis. Hemorrhagic neuroretinitis was recorded by v. Leyden. The prognosis, as a rule, is good.

Malarial Neuritis.—Tropical malaria may cause polyneuritis, but this disease is rare in this country. I have seen a number of such cases among soldiers in the tropics. Mononeuritis of the cerebral nerves has been recorded, and even optic neuritis has been mentioned. In one instance bilateral abducens palsy was noted by Strachan.

Beriberi or Kakke Neuritis.—This disease is rare in this country, although it is prevalent in the sea-coast cities of our Southern States. It is quite common in tropical countries. The etiology of beriberi is still in doubt, but it is probably caused by soil infection and not by a rice diet, although this may be a contributory cause. There are three principal forms—the atrophic, dropsical and mixed, these probably being different stages of the disease. Besides the ordinary symptoms of multiple neuritis there is great debility. Ataxia is almost always present. Dropsical effusions in nearly all of the serous cavities may be present.

Eye symptoms occur, but they have been insufficiently studied. Kessler examined the eye grounds in sixty cases and found a narrowing of the retinal arteries, whitish paling of the papillæ and, in a large number of cases, the edges of the disk were blurred. It is probable, however, that these changes in the disk are due to the general nutritional disturbance which is extreme and not to the multiple neuritis.

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[39] Die Neurologie des Auges, vol. i. 248.

Disturbances of vision, even complete blindness, may occur. I have seen this in a number of cases among the Filipinos, but this generally disappears if the patient improves. The cerebral nerves are sometimes involved, as the abducens, facial and hypoglossus.

Leprous Neuritis.—Both in the nodular and the neuritic types of leprosy we have involvement of the eyeball. In the nodular form the lepromata may invade the conjunctiva, the cornea, the anterior chamber of the eyeball, the iris, or ciliary body, or may originate in these parts. These lepromata ulcerate and finally destroy sight in both eyes.

In the neuritic type, owing to the atrophy of the facial muscles, it may be impossible to close the eyes, and there may be eversion of the lower eyelid. The upper eyelids may droop. At first there is a conjunctivitis with lachrymation, but later in the disease there will be ulceration of the cornea, and finally complete destruction of the eyeball. It is only late in the disease that such alterations in the eyeball may be expected. I have seen a large number of such cases.

Tuberculous Neuritis.—This form of multiple neuritis is rare and eye symptoms are recorded only in very few cases. Among these Fuchs records optic neuritis and nystagmus, and Rosenheim pupillary differences. The vagus, facial and phrenic nerves have been affected, but not the ocular muscles.

Syphilitic Multiple Neuritis.—According to Oppenheim, there is no doubt of the existence of a multiple neuritis due to the syphilotoxic poison, but its occurrence is doubted by many. Cases are recorded in which all of the cranial nerves with the exception of the first were involved either wholly or in part. Rudinger reported a very interesting case of polyneuritis limited to the cranial nerves which he considered as syphilitic in origin. In his case a woman, after exposure to cold, began to have paresthesia over the right side of the face and tenderness over the exit points of the right fifth nerve. This was followed by a paralysis with atrophy of the muscles of mastication of the right side, and electrical reactions of degeneration. There was also subluxation of the lower jaw and disturbance of taste in the anterior two-thirds of the tongue, and a right-sided keratitis neuroparalytica. Neuralgia of the cornea preceded the keratitis. The tear secretion in the right eye was not disturbed, neither was the sense of smell. The facial nerve was paralyzed on the opposite side and the acoustic on the same side. There were no signs of a basal meningitis or tumor, but there was a history of syphilis.

Neuritis of one facial nerve has also been reported as occurring in syphilis. It is difficult to distinguish, however, these forms from a syphilitic meningitis and the occurrence of a syphilitic neuritis alone may be well doubted.

Senile Polyneuritis.—This form of neuritis is characterized by the chronicity of its onset, the absence of sensory disturbances and of any apparent cause. This disease is rare. Schlesinger mentions a case in which there was a transient diplopia and sensory disturbances in the distribution of one fifth nerve.

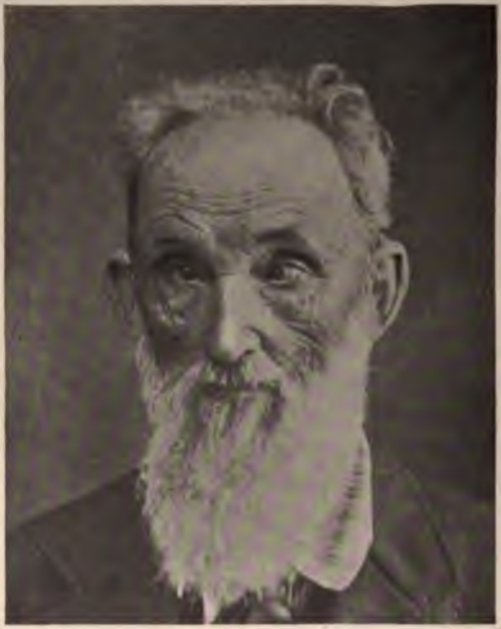

FIG. 8.—Arteriosclerotic neuritis, with paralysis of both external recti, and of the superior and inferior oblique muscles of the right eye. (Patient of Dr. C. A. Oliver.)

Von Frankl-Hochwart records an interesting case of this kind. A woman of sixty-seven, four years after a stroke of paralysis, noticed a dimness of vision in the right eye which was followed in half an hour by diplopia. A few hours later the patient could not open the right eye, but closure was normal. The eyeball was deviated outwards, the pupillary light reaction was normal, and there were no changes in the fundus. The patient died. The microscopical examination showed some disintegration of the nerve-fibres of the right third nerve, with great increase of round cells between the nerve-bundles. The vessels in the connective sheath of the nerve were engorged. The author considered it a case of arteriosclerotic multiple neuritis in which, however, only one nerve was involved.

Franceschi also recorded an instance in which there was paresis of the third, sixth and seventh nerves of one side. Microscopically diffuse areas of softening were found all through the brain. There was a parenchymatous neuritis of the involved cranial nerves. In this case there was a sclerotic condition of the arteries, this being the primary cause of senile neuritis.

With Dr. Oliver I have observed an interesting case of arteriosclerotic neuritis in a man of sixty-nine. The patient had been perfectly well up to four years ago when he first complained of diplopia. Since then he has complained of occasional headache and of a numb feeling in his extremities. The tendon reflexes were not disturbed. The pupils were equal and reacted slowly to light and in accommodation. The optic nerves were normal. There was paralysis of both external recti and of the superior and inferior oblique muscles of the right eye. His arteries were markedly sclerotic. There was no history of syphilis.

There can be no doubt of the occasional occurrence of ocular palsies due to the neuritis caused by the pressure of the sclerosed basal arteries. This is perhaps not sufficiently recognized as a causal factor. (See chapter by Dr. Hirsch.) Pathologically, this condition is sometimes observed. Recently I found both optic nerves much degenerated, and of crescent shape, due to the pressure of the sclerosed anterior cerebral arteries.

Carcinomatous Multiple Neuritis.—This form of polyneuritis may be due first to an autointoxication, second to a carcinomatous infiltration of the sheaths of the peripheral nerves, and, third, to an acute general carcinomatous infiltration of the peripheral nerve endings.

The character of the multiple neuritis due to carcinomatous intoxication does not differ from the other forms of polyneuritis due to toxic causes. In a case of Miura's there was a slight optic neuritis with an absolute central scotoma. There was also nystagmus on lateral and upward deviation and diplopia. These symptoms appeared several weeks before death and persisted. The microscopical examination by Uhthoff failed to show any cause for the optic neuritis. The various peripheral nerves, as well as the orbital parts of the third and sixth nerves, showed degeneration. The spinal cord and the different cranial nuclei were normal. The slight swelling of the papillæ was attributed to the broadening of the nerve-fibres in the upper layers.

Dr. Spiller and I have recorded a remarkable case of cerebral involvement due to a cancer. A woman who had a recurrent carcinoma of the breast five weeks before death complained of failing vision in both eyes, more marked in the left. The right pupil was smaller than the left and responded to light and in accommodation, but the left pupil did not. There was some exophthalmos of the right eye. The external rectus muscle of each side was paralyzed and there was, besides, a paralysis of the left superior rectus muscle, and ptosis on the same side. Unfortunately no ophthalmoscopic examination was made. An examination several days before death showed an almost complete internal and external ophthalmoplegia of the left eye, and a distinct, but not equal, weakness of the left seventh nerve. There was some headache and dizziness, weakness of the limbs and absent knee-jerks.

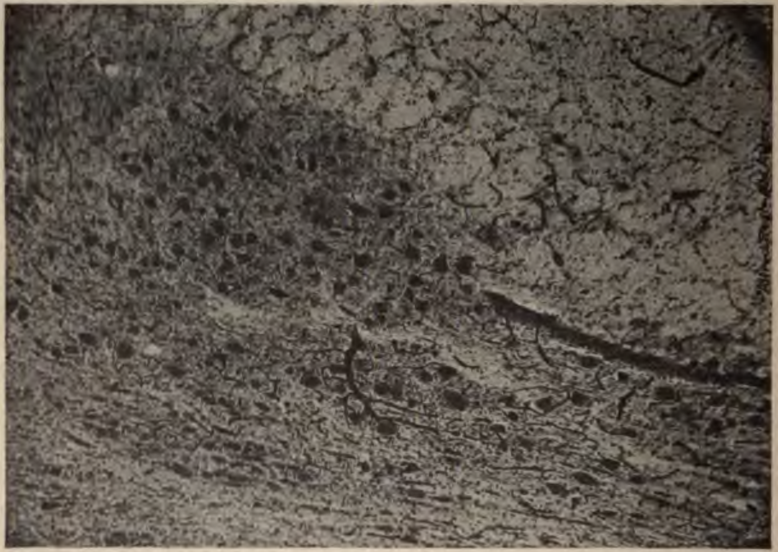

FIG. 9.—Case of carcinomatous polyneuritis. Illustration shows marked congestion of the blood-vessels and disease of the oculomotor nuclei.

The macroscopical examination did not show anything. Microscopical examination showed the optic nerves to be normal. Some of the axis cylinders of both third nerves, of the right fourth, of the right sixth, and of both seventh nerves, were distinctly swollen, but this was especially marked in the right sixth nerve. The left fourth nerve was normal. The left sixth nerve was not obtained. Some of the nerve cells of both oculomotor nuclei were much diseased, and the nucleus of each abducens nerve was intensely diseased. The other cranial nuclei, as well as the cortical cells, were normal. The blood-vessels all through the brain were intensely congested,—this being especially marked in the cerebral peduncles and the posterior part of the pons.

This case illustrates well the diffuse action of the carcinoma toxin. It is also noticeable that the pathological findings are not commensurate with the symptoms, for microscopical examination may denote a degeneration of a cranial nerve, the clinical symptoms of which may have been entirely absent and *vice versa*.

In the second form of polyneuritis, that due to an infiltration of cancer-cells in the nerve sheaths, we may have, as in the case of Saenger, an infiltration of cancer-cells in the pia covering the nerve-fibres at their exit from the base of the brain. Saenger also found an infiltration of cancer-cells in the pial covering of the cortex.

Raymond has very recently called attention to a third form of cancerous polyneuritis, the acute, general, miliary form. The symptom complex is similar to the other forms of polyneuritis. Macroscopically no changes can be found centrally or in the peripheral nerves, but microscopic examination shows multiple lesions in the finest intramuscular nerve endings, these being due to the compression by the innumerable cancerous emboli in the muscles, a true carcinomatous neuromyositis. No instance is recorded, however, in which the cranial nerves were diseased.

Diabetic Neuritis.—It is recognized that diabetes may cause a disease of the peripheral nerves. It is, however, improbable that the occasional involvement of a cranial nerve is part of a peripheral neuritis, for when such symptoms occur, we probably have a general toxic process due to the diabetic poison. This subject is discussed in another chapter.

Primary Neurotic Atrophy.—Because of our lack of sufficient pathological evidence, confusion still exists as to the cause of this disease. It occurs in families and is distinguished by atrophy, developing in the distal portions of the extremities. The reflexes are absent and there are few sensory disturbances. Pathologically, in the few cases recorded, alterations have been found in the peripheral nerves and muscles, and the nerve-cells of the anterior horns of the spinal cord.

Optic atrophy accompanying this disease has been recorded only by Ballet, Rose, Vizioli, and A. Gordon. In a case under my observation in the Philadelphia Hospital the patient gave a distinct history of paralysis of the left external rectus and the left superior oblique muscles. The diplopia persisted for some time, but has disappeared. There was no history of syphilis.

It is difficult to explain the occurrence of the optic atrophy and the involvement of the ocular muscles. It is probable, however, that we have here conditions foreign to the disease itself.

CHAPTER XVI.

NEUROSES AND PSYCHOSES.

By G. E. de SCHWEINITZ, A.M., M.D.

OCULAR MANIFESTATIONS OF HYSTERIA.

The investigation of the disturbances of visual sensation to which hysterics are subject has acquired much importance, and the ocular stigmata of hysteria, as Parinaud has said, constitute a symptomatology which is distinct and in some phases unvarying. The following somewhat artificial subdivisions of the subject are convenient:

1. Complete anæsthesia of the visual sense, or hysterical amaurosis.
2. Incomplete anæsthesia of the visual sense, or hysterical amblyopia.
3. Defective and painful visual sensation, or hysterical asthenopia.
4. Anomalies of accommodation, or hysterical cyclospasm and cycloplegia.
5. Anomalies of the iris movements, or hysterical pupil phenomena.
6. Anomalies of the eyelid and eye-muscle movements, or hysterical contractures, pseudopalsies, and palsies of the palpebral and bulbar musculature.
7. Previously unclassified hysterical ocular phenomena.

I. COMPLETE ANÆSTHESIA OF THE VISUAL SENSE, OR HYSTERICAL AMAUROSIS.

Hysterical Amaurosis.[1]—This term is applied to a condition characterized by complete abeyance of visual sensation. It occurs both as a unilateral and a bilateral affection.

(*a*) **Bilateral Hysterical Amaurosis.**—The literature has been collected and analyzed by Cruchet,[2] Kron,[3] and Kernéis.[4] The last author succeeded in gathering 34 cases, and since the publication of his thesis a number of additional examples have been reported,—for instance, by

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[1] For the historical side of this question and for the earlier observations the reader is referred to Gilles de la Tourette's Traité Clinique et Therapeutique de l'Hystérie, 1891, p. 321; also to P. Pansier's Les Manifestations Oculaires de l'Hystérique, Paris, 1892.

[2] Archiv. de Neurologie, 2d Series, xii., 1901, p. 177.

[3] Neurologisches Centralblatt. xxi. 1902, p. 584.

[4] Etude sur la Cécité hystérique ou Amaurose Hystérique totale et bilatérale, Bordeaux, 1902.

Vetter, McReynolds, Dupuy-Dutemps, Le Roux,[5] Hirsch,[6] and Cabannes.[7]

In most instances the loss of sight has been absolute, or, form-sense and color-sense being lost, the sensation of bright light is still retained, —that is, the patient has subjective sensations of light. The onset may be sudden or gradual. Although the conditions necessary to create double hysterical blindness usually follow an hysterical attack and the affection has been described by Binswanger as an interparoxysmal, morbid state, this is not necessarily the case, as the amaurosis has been encountered without any connection with an attack,—for example, after a surgical operation, or, again, as in Leroux's patient, without any preceding hysterical phenomena. Usually both eyes are attacked simultaneously; sometimes one eye may be affected later than the other,—for instance, after a few hours, or even after some months. More women than men have been affected (4 men to 22 women, according to Kron). The age in women has varied from 10 to 26 years; in men from 22 to 37 years. The pupils may be dilated or contracted. Generally they react to the influence of light; sometimes only a feeble contraction follows the light stimulus; occasionally the pupils are dilated and insensitive to light (Kernéis). Accompanying the amaurosis the following symptoms have been observed in some of the cases: Lagophthalmos, ptosis, spasm of the orbicularis, anæsthesia of the conjunctiva, nystagmus, conjugate deviation, and convergent strabismus.

To establish the diagnosis of hysterical amaurosis there must be excluded: Ocular disease sufficiently great to produce blindness, congenital amblyopia with high grades of refractive error, and particularly certain forms of so-called retrobulbar neuritis. (For further discussion of this, see page 638). Evidently complete blindness, in the absence of ophthalmoscopic signs of optic nerve disease, associated with normal pupil reaction to light impulses, would strongly suggest, if it did not prove, the presence of the hysterical taint, but because in rare instances, temporarily at least, the pupils under these conditions fail to react to light, the absence of the pupil reflex does not necessarily exclude hysteria.

All cases on record have recovered vision sooner or later, and therefore the prognosis *quoad visum* is good. Indeed, usually when the amaurosis disappears, it does so, as Pitres remarks, without leaving a trace. The duration of the blindness, however, has varied considerably. Following Kron's suggestion, the cases may be divided into: (*a*) The transitory forms, lasting from a few hours to several days; (*b*) the short-enduring forms, lasting from two to six weeks; and (*c*) the long-enduring forms, lasting from four months to ten years. To these should be added the *intermittent varieties* described by Koenigstein, in which the blindness appears at certain definite hours of each day, and lasts for a time and then disappears. According to Kernéis, if the blindness has appeared suddenly, it will disappear suddenly; if it has arisen during a crisis, it will be during a crisis that it disappears; if it is without a crisis that the blindness has appeared suddenly, it will be equally sudden and without a crisis that the patient will recover his sight; if it has made its appearance gradually, its disappearance will be a gradual one. As Kernéis himself remarks, these rules meet with exceptions. The blindness may be exceedingly stubborn and last, as has already been noted, for years, even for ten years, as in Harlan's well-known case. Before the entire disappearance of the blindness, there may be a stage of monocular amaurosis, as in McReynolds's case, with recurrences, and occasionally a period of temporary hemianopsia. (See also page 636.) The first color seen by the patient on recovery from the blindness is red. (Cruchet.)

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[5] Archives d'Ophthalmologie, December, 1903, p. 801.

[6] Prag. med. Wochenschr., 1904, xii. 288.

[7] Gaz. hebd. d. sc. méd de Bordeaux, 1905, xxvi. 3.

Certain writers are sceptical in regard to the long-enduring forms of hysterical blindness,—that is to say, sceptical in regard to the hysterical nature of the affection. For example, B. Sachs[8] writes: "I would prefer not to accept as a fact the occurrence of complete hysterical amaurosis of months' duration until more convincing cases shall have been brought forward." Similar doubts were entertained by Mauthner, and are implied by Schmidt-Rimpler.[9] I have never examined a patient with bilateral hysterical amaurosis, except as it occurs in the most temporary way during an attack, and can reply to these criticisms only by saying that some of the cases appear to me to be above suspicion,—for example, Mendel's case, which Sachs himself quotes.

(*b*) **Unilateral Hysterical Amaurosis.**—Unilateral hysterical amaurosis, in which there is complete abeyance of the visual sensation of one eye, it is usually stated, is a more commonly observed phenomenon than the bilateral variety of the affection. According to Kron, however, more instances of the double than of the single amaurosis are on record, —26 compared with 23. Doubtless the explanation of this is that all, or nearly all, of the cases of double amaurosis have been recorded, while only a comparatively few unilateral instances have been published. Either eye may be affected, and, according to Briquet, one eye is not more frequently selected than its fellow, but in Kron's collection the right eye was blind 13 times and the left 9 times. Females are more liable than males, in the proportion of 2 to 1. The affection is not confined to adults, and pure examples have been observed in children,—for instance, by Veasey in a child of 11. I have recorded one case in a negro child of 11 and seen another in a girl of 14. The difficulty of diagnosis in very young children is apparent.

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[8] Journal of Nervous and Mental Diseases, xxvi. 1899, p. 347.

[9] Die Erkrankungen des Auges im Zusammenhang mit anderen Krankheiten, Wien, 1898, p. 256.

Unilateral hysterical amaurosis may arise suddenly as the result of emotional excitement, fright, trifling injury, menstrual pain, etc., or occur as the termination of an hysterical attack, and return with the ending of the second attack, constituting, like the bilateral affection, the interparoxysmal morbid ocular condition, or, according to Binswanger, it may exist for a long time without any connection with attacks or emotional disturbance. The period of blindness, like the bilateral variety, may be brief, or last for years. One of Pitres's patients, a child aged 10, was blind four years. In true unilateral hysterical amaurosis the opposite eye possesses normal vision and visual field, or even a visual field more extended than that which is ordinarily considered normal (Parinaud, Morax). More often the opposite eye exhibits defective visual acuity, contracted visual field, etc., symptoms which will presently be elaborated (page 621). Under these circumstances there is really bilateral disturbance of the visual sensations, —that is, blindnes, or amaurosis, on one side, and reduced vision, or amblyopia, on the other. Associated with the amaurosis there may be other disturbances of sensation—anæsthesia of the skin, perhaps confined to one side,—that is hemianæsthesia, anæsthesia of the mucosa of the pharynx, of the conjunctiva, and of the cornea. The other ocular conditions previously noted with the bilateral form of the affection may also exist. While the amaurotic eye is likely to be on the side of the greater cutaneous anæsthesia, this rule is not an invariable one. (See Page 113.)

The Effect of Binocular Fixation on Monocular Amaurosis.—Schweigger, Bernheim, Pitres, Janet, and other observers have noticed and described the disappearance of a monocular amaurosis during binocular fixation, and we are especially indebted to Parinaud[10] for a study of this subject. Briefly, some hysterics completely amaurotic in one eye lose this blindness during binocular fixation, as may be proved by ordinary prismatic, stereoscopic, or other tests. Thus, if before the admittedly sound eye of a patient completely hysterically amaurotic in the other eye a prism is placed through which a test-object is regarded, and two images are seen, there is evidence that the blind eye has been associated with the other eye in the act of binocular fixation. For example, if previous examination has shown that the right eye is amaurotic in monocular fixation, while with a prism base down before the left eye the patient sees two images, and on closing the right eye the lower image disappears, it is evident that the blind eye has participated in the act of binocular vision.

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[10] The Ocular Manifestations of Hysteria, System of Diseases of the Eye, edited by Norris and Oliver, vol. iv. 1900, p. 741; and Annales d'Oculistique, cxxiv. 1900, p. 17.

According to Parinaud, as well as other observers,—for example, Gilles de la Tourette,—the re-establishment of vision in the amaurotic eye at the moment of binocular fixation is concerned only with the central perception. The peripheral portion of the visual field remains abolished, both in monocular and binocular vision. Sometimes experiments fail to elicit the reappearance of sight in the amaurotic eye in the act of binocular vision, either because the defective sight is too great, or perhaps because the patient never possessed, or possessed only in imperfect degree, binocular vision.

Cruchet[11] describes an amaurotic hysterical patient who did not have binocular vision, and who with all the tests answered as if she was really blind in one eye. According to him the amaurosis should be considered in two stages,—the first one, in which there is complete abolition of binocular vision, and the second one, more frequently observed than the other, in which there is intermittent and generally perverted conservation of binocular vision. He states that in cases of unilateral hysterical blindness, monocular vision exclusively by the good eye is the rule; but at certain moments and under certain conditions the monocular vision of the sound eye becomes monocular vision of the amblyopic eye,—that is, there is alternation. Again, monocular vision may seem to become binocular, but only apparently. Really it is simultaneous vision,—that is, fusion of the images does not take place. Sometimes, the monocular vision becomes binocular vision, with fusion of certain images and not of others; the binocular vision, to use the expression of Antonelli,[12] is dissociated. Finally, at certain moments and under certain conditions monocular vision really becomes binocular with complete fusion of all the images, a condition which Cruchet regards as essentially temporary in character.

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[11] Archiv de Neurologie. 2d Series, xii. 1901, p. 177.

[12] Archives d'Ophthalmologie, xvii. 1897, p. 218.

Concerning the Nature of Hysterical Amaurosis.—It may be stated in the light of our knowledge that hysterical blindness, such as has been described in the preceding paragraphs, is not due to any failure of the retina to appreciate the images of external objects, but to a functional disturbance of cortical centres. A patient with monocular amaurosis of hysterical nature sees with the blind eye, as may be determined by the ordinary tests which are used to detect pretended blindness.

Again, Binet and Janet have shown that if before the eye of an amaurotic hysteric some object is held and the patient is afterwards hypnotized, he will be able to tell that which was previously exhibited and which he regarded with an apparently blind eye. Evidently the patient really saw the object. As Prince puts it, "the hysterically blind see, but their visual sensations are not in connection with their dominating waking consciousness. There would seem to be a shutting off from the field of consciousness of certain perceptions which form a group of segmented sensations, more or less by themselves, and which by artificial means,—for example, the hypnotic state,—would as memories be brought into relation with the remainder of the conscious field."

Because hysterics apparently blind in one eye may by tests ordinarily applied to malingerers be proven to see with this eye, they have been characterized as cheats; but, as Pitres points out, the uniformity of answers given by the subjects of unilateral hysterical amaurosis cannot be reconciled with the theory of deception in the gross sense of this term.

According to George C. Harlan, patients with hysterical blindness may be divided into three classes: those who deliberately simulate the blindness, those who see unconsciously but are not capable of conscious vision, and those who are really transiently absolutely blind.

Binswanger[13] quotes Binet's conclusion that the disturbances which lie at the bottom of hysterical anæsthesia, no matter what its nature may be, must have their seat in the cerebral cortex, because in order that a peripheral stimulus may produce not only movements but also concepts, it must have reached the centres in which concepts are formed,—that is, the highest composite centres of the cerebrum. Bernheim believes that an amblyopic hysterical subject with the aid of imagination unconsciously neutralizes the visual image which has been perceived in a normal manner, although, as has been shown, and as Pitres points out, the patient does not neutralize binocular but apparently only monocular perceptions. Janet, however, from his experiments leans to the theory of Bernheim, and thinks that it demonstrates the psychic or cortical nature of this sensory disturbance, while Charcot rejects Bernheim's explanation that hysterical blindness is exclusively a psychic phenomenon,—that is, an illusion of the mind and not a true sensory anæsthesia.

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[13] Specielle Pathologie und Therapie. H. Nothnagel, xii, Band, i. Hälfte, ii. Abth., Die Hysterie, von Prof. Dr. Otto Binswanger, Wien, 1904, p. 979.

Referring again to the interesting fact that in a certain number of instances, at least, monocular hysterical amaurosis disappears in binocular fixation, we find that Parinaud[14] dismisses Bernheim's theory that such phenomena represent a blindness of the imagination due to the destruction of the image by a psychic agent, with the curt expression that "he does not understand such language," and thinks that the interpretation of hysterical visual symptoms depends upon the belief in the existence of certain relations between the retina and the visual centres in monocular and binocular sight, as well as definite and distinct relations in central and in peripheral vision. He goes on to say, "If we admit the crossed relation between each eye and the opposite hemisphere in monocular vision, the left eye will become amaurotic when the right side of the brain is affected; but if in binocular vision both eyes are connected with a single hemisphere, it seems clear that if that hemisphere be the left and healthier one, the left and amaurotic eye will be able to see."

Charcot, quoted by Binswanger, has demonstrated that hemianæsthetic patients with monocular color blindness are able to identify the color accurately during binocular vision, even with the achromatoptic eye, and therefore favors the view that there are functional disturbances in the intracerebral centres for monocular vision, those for binocular vision being uninjured. Similar observations have been made by Parinaud. I shall return to this subject in a later paragraph.

While there is no proof of it, so far as I am aware, it is conceivable that some toxin is liberated during an hysterical attack and the retina poisoned, or perhaps the cortical centres, by this toxin, producing, according to its virulence, a temporary, an enduring, or a long-continued blindness, exactly as this occurs in uræmia, in diabetes, and in certain intestinal fermentations. It would be confessedly difficult to reconcile this theory with unilateral amblyopia, unless one were willing to admit a selective action of the toxin upon one retina, exactly as in very rare toxic blindness,—for example, quinine blindness,—is unilateral.

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[14] System of Diseases of the Eye, edited by Norris and Oliver, iv. p. 743.

II. INCOMPLETE ANÆSTHESIA OF THE VISUAL SENSE, OR HYSTERICAL AMBLYOPIA.

This term is applied to a number of different visual disturbances, of which the most important are: reduction of visual acuity, contraction of the visual field, disturbance of the color and light sense, and anomalies of accommodation. In contradistinction to the affections which have been described in the preceding paragraphs and which represent a total loss of visual sense, either unilateral or bilateral, these are incomplete disturbances of sight. They represent, in the language of Binswanger, the incomplete anæsthesias; "they are polymorphous and composite in character, both as regards their extent and as regards the special variety of the visual disturbance."

(1) **Reduction of Visual Acuity.**—By this is meant a failure of the affected eye, or eyes, to obtain full visual acuity in the absence of refractive defects and pathological changes, as not infrequently occurs in hysteric patients.

(2) **Disturbances of the Visual Field in Hysteria.**—(*a*) **Visual Field for White.**—Many of the records of the visual field examinations in hysterics and neurasthenics are untrustworthy because no definite information is given in regard to the light, the size of the test-object, character of the refraction, the length of time employed in taking the visual field, the distance of the examined eye from the fixation point, etc. Unfortunately, there has been no uniform method, and hence the comparison between the results of different observers is often difficult and probably accounts for the discrepancy in statement. Thus, one observer, in taking the visual field, moves the test-object from the periphery towards the centre and waits until the patient distinctly sees the object, and then records its position on the perimeter arc; while another evidently has recorded the points at which the patient first becomes conscious of some object,—for example, a square of white or colored paper on the perimeter arc. There is almost a uniform absence of statement in regard to the patient's refraction under these conditions, and very rarely have control examinations been made with the campimeter, or, except in recent times, with the visual field taken at varying fixation distances. (For a further consideration of this subject, see Chapter IV.)

Just as a certain number of hysterics have normal acuity of vision, they may also have a normal visual field for white. Pansier[15] states that although the visual field for white may be normal, he has found a true enlargement of the color field, with or without inversion of the color circles, in all non-amblyopic hysterics which he has examined. On the other hand, Frankl-Hochwart and A. Topolanski[16] maintain that in hysteria with normal sensation there is usually no contraction of the visual field, and von Reuss[17] has also noted that hysteria may exist without any changes in the fields of vision. John K. Mitchell and myself[18] have observed a completely normal character of the visual field in a certain number of hysterics, or simply contraction of the color fields with unaffected form fields.

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[15] Les Manifestations oculaires de l'Hystérie, Paris, 1892, p. 17.

The essential characteristic of the visual field in hysteria is *concentric contraction*, if this is measured with a white test-object on a black background. It represents, to use the language of Parinaud, "an insensibility of a portion of the field to white light, and this insensibility develops from the circumference to the centre in such a way that a graphic drawing of the contraction would be represented by an almost circular line." Naturally, as Parinaud further maintains, *concentric* must not be taken in a too literal sense of the term, because, although quite noticeable on the temporal side where the field is normally largest, it may be most restricted on the nasal side or above. Furthermore, this visual field should manifest its contraction at the very beginning of the measurement, as von Reuss[19] insists, and not be called into existence by fatigue during examination.

The amount of the reduction of the volume of the visual field varies from slight contraction, most noticeable on the temporal side, to such an extreme restriction that the most peripheral circle is just beyond the fixing point. Binswanger is willing to apply the term "pronounced" only to those cases in which the normal visual field is reduced fully one-half. Should the patient have possessed originally a minimal or small physiological field, slight reduction, if bilateral, would be difficult to determine unless one should find, as Parinaud suggests, a concomitant hysterical disturbance of accommodation. (See page 28.) Binswanger has never observed "the gravest cases, in which the visual field is reduced to the fixation point," and he therefore differs from Parinaud, Gilles de la Tourette, and Janet. I have on a number of occasions seen extreme reduction in the size of the visual field (see Fig. 1), and can in this respect confirm entirely the observations of the French observers concerning which Binswanger is sceptical.

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[16] Deutschmann's Beiträge zur Augenheilkunde, ii. 1895, p. 46.

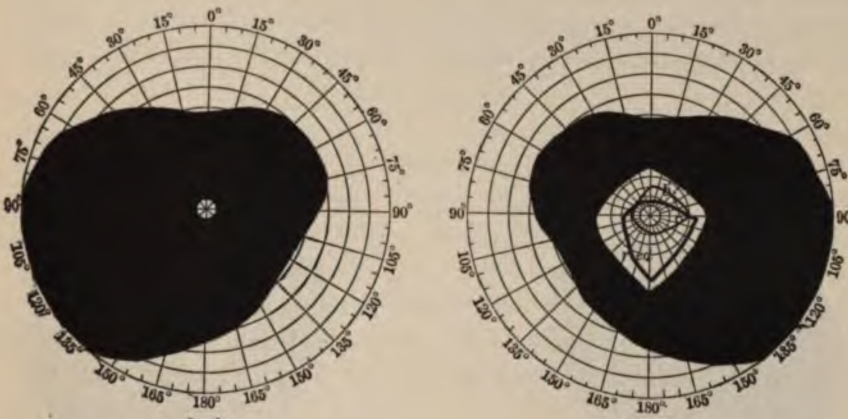
[17] Das Gesichtsfeld bei functionellen Nervenleiden, Wien, 1892.

[18] Journal of Nervous and Mental Diseases, January, 1894.

[19] Loc. cit., p. 52.

The hysterical field contraction is of long endurance, lasting for days, months, and even years, and the variations in size which it undergoes, except those which are later recorded, are comparatively unimportant until recovery occurs, when the visual field again may assume its normal size.

The behavior of the contracted visual field, when it is measured with the point of fixation placed at varying distances from the observed eye, is of importance. Schmidt-Rimpler[20] maintains that each perimetrically recorded concentric contraction of the visual field should also be investigated with a campimeter stationed at varying distances from the patient, and Wollenberg, quoted by Binswanger, found under these conditions that in the majority of cases, although not always, as the distance between the patient and the blackboard is increased, the size of the visual field is enlarged. On the other hand, Richard Greeff[21] states that the contracted visual field maintains the same size, no matter at what distance the point of fixation of the examined eye is placed,—in other words, that the field has a *tubular character;* the hysteric has, to use the term which Greeff has given, a "röhrenförmiges Gesichtsfeld." The examination is made as follows: The visual field is measured, as usual, with a perimeter, and if it is found contracted in decided degree,



Left *Right*

FIG. 1.—Grand hysteria, scattered anæsthesia. Woman, aged 26; patient of Dr. J. K. Mitchell. Contracted tubular fields; partial reversal right; all color sense absent in left field.

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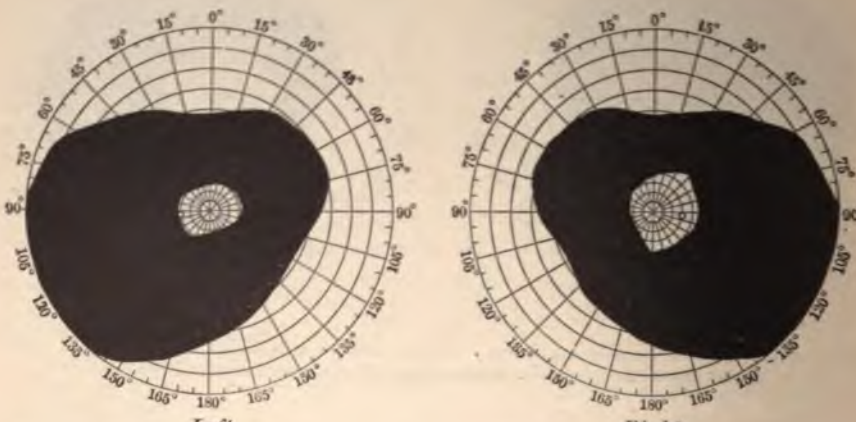
[20] Deutsch. med. Wochenschr., 1892, Nr. 24.

[21] Berlin klin. Wochenschr., May 26, 1902, p. 496.

the point of fixation for the examined eye is removed one metre distant and the measurements repeated. Instead of an increase in the width of the field, it may remain just as narrow as it was prior to the lengthening of the fixation distance, and the same is true if this is still further increased,—for example, to two, three, or five metres. That this observation is entirely correct I am able to confirm as the result of many examinations. (See Fig. 2.)

According to Greeff, the cause of the tubular field is connected with the underlying nature of the disease; that is, in hysterical patients a definite conception frequently dominates the sensorium, and this applies also to the visual field. Von Hoesslin is unwilling to admit that symptoms which are physically impossible are genuine signs of disease, and he regards the person who exhibits the tubular field phenomenon as a malingerer, and if hysteria is present, then the subject is a hysterical malingerer. Arnheim and other observers are not in accord with von Hoesslin in this contention, and I agree with them and with Wilbrand, that "we have no right to cast suspicion of malingering on a patient unless we are able to convict him or her of malingering."



Left *Right*

FIG. 2.—Hysterical girl. Patient of Dr. J. William White. Typical tubular contracted field; no change in size at 30 Cm., 1, 2, 3, and 4 metres.

Although the contraction of the visual field may be extreme, visual acuity may be normal; on the other hand it may be reduced. Inasmuch as in many cases the peripheral vision is completely abolished, producing typical tubular vision, if central sight is affected then amblyopia may give place to amaurosis and the condition of complete hysterical blindness. It should be remembered, however, that such amaurosis need not have—indeed, does not usually have—concentric visual field contraction as its prodrome, but appears suddenly.

The contraction may be unilateral (8 times among 79 cases examined by Parinaud) or bilateral, generally the latter. Usually, as Féré, Parinaud, Landolt, Wilbrand, Saenger, and others have shown, it is associated with disturbances of cutaneous anæsthesia, being, when these are present, in the language of von Frankl-Hochwart and Topolanski, a fairly constant phenomenon. In bilateral cases the restriction of the visual field is usually greater on the hemianæsthetic side, or upon that side which exhibits the greater surface or degree of anæsthesia; rarely the opposite eye is the more affected. Unilateral contraction of the visual field commonly appears on the hemianæsthetic side. It should be remembered, however, that the presence of cutaneous anæsthesia does not necessarily imply the presence of restricted visual fields, nor is it necessary that they should appear, as J. K. Mitchell and myself have shown, when the general symptoms of hysteria are of the highest grade.

According to Gilles de la Tourette,[22] concentric narrowing of the visual field is the most frequent ocular stigma in hysterical subjects, and Dana says that it is the commonest of all forms of anæsthesia. Pitres remarks that it is one of the most frequent sensory disturbances of the great neurosis and one of the best stigmata of hysteria. Binswanger,[23] on the other hand, writes, "The scepticism with which we regard the statements of Charcot's school (Galezowski, Parinaud, Landolt, Féré, and others) increases in direct proportion to the growth of our experience in this subject; for these investigators attribute too much importance to the concentric contraction of the visual field in hysteria." The value of this symptom seems to me exceedingly great, although J. K. Mitchell and myself were unable to find it as frequently among the hysterics whom we examined as would appear to be the case in the French clinics.

It has already been pointed out that the contracted visual field is one that appears at once and not as the result of repeated examination, and that in a certain sense it is a permanent stigma, or perhaps, more accurately, an enduring one, differing in this respect from the one which will later be described in connection with neurasthenic cases. It is, however, subject to certain variations, which must now be mentioned. They are these:

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[22] Traité Clinique et Therapeûtique de l'Hystérie, 1891.

[23] Loc. cit., p. 209.

In unilateral visual field contraction the narrowed field may be made to disappear by irritating the skin, and to develop in the healthy eye; in short, a transfer takes place. It may be transferred to the other eye by suggestion. Also, as Parinaud has shown, if the unaffected eye be opened and the visual field previously contracted remeasured, it may increase its extent. Referring to this fact, Janet [24] suggests that under these circumstances the sensations of the sound eye bring into personal perception by a pre-established association those of the neglected eye. The extent of the field is furthermore modified by spherical glasses, by tinted glasses, and by the action of mydriatics, as has been well shown by Parinaud, Förster, and as I have frequently demonstrated. A visual field contracted for a white test-object may reach greater or even normal extent if investigated with a bright electric light.

The visual field may be diminished in size by mental condition, by physical depression, preoccupation, emotions, fixed ideas; in general terms, as Janet puts it, in a great number of hysterics the effort of visual attention contracts in a very notable way their visual fields. The variations in perimetric examinations are not necessarily dependent upon simple retinal fatigue, but, as Schmidt-Rimpler, Schultze, and others have shown, may depend upon a diminution of the cortical, psychical functions, and be due to damage of the power of concentration.

Binswanger emphasizes the importance of the psychic condition at the time of examination and its effect on the visual field, and insists that patients should be methodically investigated at different times with due regard, not only to their power of concentration, but also to their emotional state, and to the presence of definite disturbances of the intellect, and, finally, to the relation of their condition to antecedent hysterical attacks.

It has been the custom of some authors to classify with hysterical fields "*the shifting type*," originally described by Förster in cases of anæsthesia of the retina, in which the visual fields differ according as the examination is conducted from the temporal to the nasal, or from the nasal to the temporal side, the contraction being pronounced on the nasal side in the former and on the temporal side in the latter; the *exhaustion type* of Wilbrand, in which the test-object is moved from the temporal to the nasal and from the nasal to the temporal side several times in succession, across the entire width of the perimeter, indeed, as often as the field continues to diminish; *unstable concentric*

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[24] The Mental State of Hysterics. Translated by C. R. Corson. G. P. Putnam & Sons, 1901, p. 69.

limitation,—that is, a field which is constantly changing during the examination; the *exhaustion-spiral type,* in which the tracing of the visual field appears coiled like a watch-spring in consequence of its limits becoming concentrically smaller; and finally, the *recuperation-extension type,* in which the restricted field may extend during rest, or by a strong effort of will. These five types, classified and elaborated by von Reuss,[25] do not, however, belong strictly to pure hysterical cases, and will be considered in more detail in the section devoted to neurasthenia.

One form of field, however, von Reuss feels should be classified with those which are more strictly hysterical in type, namely, the so-called "*oscillating field,*" described by Wilbrand and O. Koenig, in which the object disappears and reappears several times in the same meridian, and in which a similar oscillation applies to color. This phenomenon has been ascribed to rapid change in the sensitiveness of the retina. (Fig. 3.)

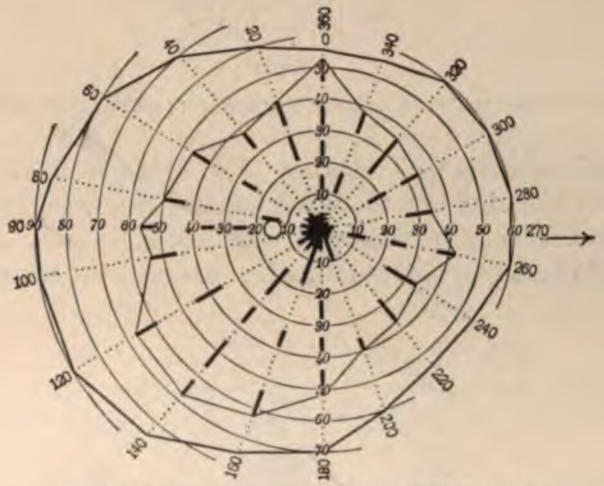

FIG. 3.—Left field. Oscillating field. (After Wilbrand.)

As Wilbrand has shown, if the areas in the visual field in which the test-object disappears are studied in association with one another, they assume a *zonular form,*—in other words, an *annular scotoma* results. Such scotomas as hysterical manifestations have been described by a number of observers,—for example, Ziehen and Casey A. Wood. (Fig. 4.) The latter author, although unwilling to rely upon this sign alone in the diagnosis of hysteria, has observed it when other ocular indications of this neurosis were wanting. Von Reuss maintains that there may be a double-ring scotoma, a fragmentary ring scotoma, or that the oscillating field may be associated with central scotoma, and, as in a case of Wilbrand's, with scintillating scotoma, or temporary hemianopsia. A certain degree of stability characterizes these scotomas which distinguishes them from exhaustion cases. According to Wilbrand, *a central exhaustion scotoma* is rare. I have observed an absolute central scotoma in a hysterical woman which lasted for weeks (Fig. 5).

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[25] Loc. cit. See also a good review of von Reuss's book by J. J. Evans, Ophthalmic Review, xxi. 1902, p. 339.

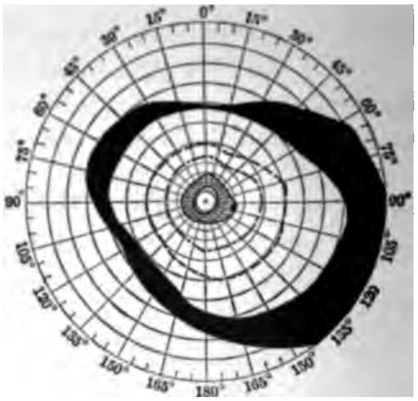


FIG. 4.—Annular scotoma in hysteria; moderate contraction of form field; no reversal of color lines.

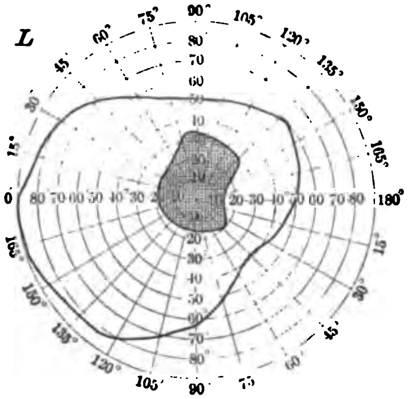
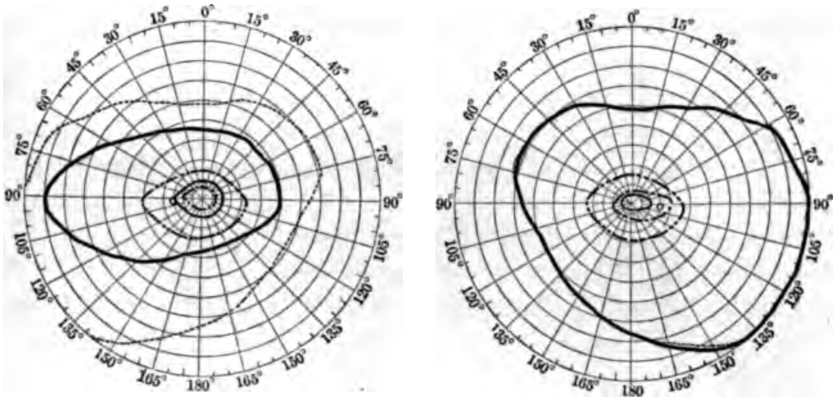


FIG. 5.—Central scotoma in hysteria.

(*b*) **Visual Field for Colors.**—Under this heading must be considered concentric contraction of the color fields, dyschromatopsia and acromatopsia, and certain rare conditions to which the term hyperchromatopsia may be given.

Just as there may be a narrowing of the visual field due to an insensibility to white light, and which, in the language of Parinaud, constitutes the fundamental condition of this type of hysterical amblyopia, so also there may be an insensibility to colors, which begins at the periphery of the visual field and occasions a contraction of the color field. The color circles may be found within the narrowed circle for white exactly in their physiological order,—that is, from without inward, blue, yellow, red, green, and violet,—the entire restricted field is really a much reduced normal visual field.



Left *Right*

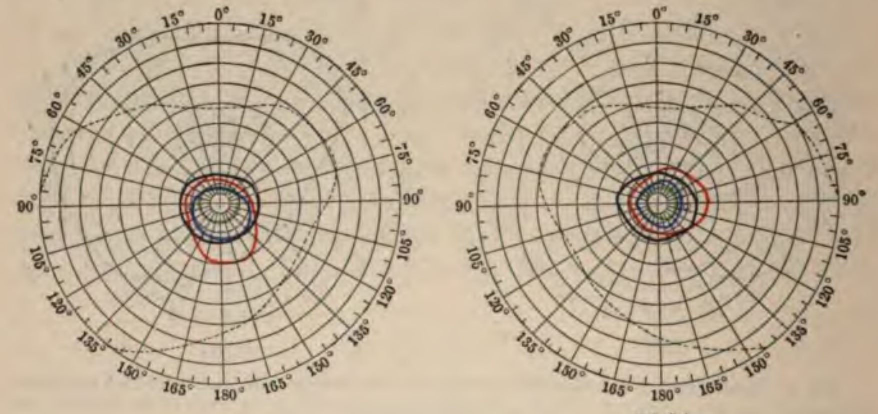
FIG. 6.—Right hysterical hemianæsthesia; patient of Dr. Wharton Sinkler. Partial left anæsthesia reversal of color lines; outer circle, white, next inner, red, then blue and green; form field not contracted, R. E.; moderate contraction, L. E.

Sometimes, as Nonne, J. K. Mitchell, myself, and others have shown, the restriction is confined to the color fields, while the white field remains unaffected (Fig. 6, right). As long as the color fields, even though restricted, retain their normal physiological relation one to the other, such restriction, as Parinaud insists, does not necessarily indicate dyschromatopsia, but might depend on individual differences found in the normal state, or on the light and the objects employed, and upon the narrowing due to insensitiveness to white light.

Under normal circumstances the color fields when tested with saturated colors in good illumination occur from the periphery to the centre in the following order: blue, yellow, red, green, violet. Now in hysterical patients a somewhat characteristic variation is that the red field is the last to be affected, with the result that its extent may equal that of blue, so that these color lines coincide or cross one another, or it may exceed it and become the most peripheral of the color circles.

This is the so-called *inversion of the color field*, and represents much more certainly, again to quote Parinaud, the presence of dyschromatopsia than a mere limitation of the extent of the visual field for colors. More than this, the red field may exceed in extent not only the blue field as described, but that for white, and become not merely the peripheral color circle, but the peripheral circle for the entire field.

Binswanger states that in his clinic he has observed cases in which the visual field for red was extraordinarily diminished, so that it was exceeded by the visual field for green; similar observations have been recorded by v. Hochwart and Topolanski and Wilder, and Pansier has seen the green circle become the most peripheral one. Such exceptions, however, do not destroy the main fact, that hysterical subjects have a predilection for red, constituting a characteristic of this type of dyschromatopsia (Fig. 7).



Left *Right*

FIG. 7.—Hystero-catalepsy; woman aged 20; patient of Dr. S. Weir Mitchell. Typical concentric contraction and reversal of blue and red lines. In some meridians the red field exceeds the white field.

Coming to the question of partial abolition of the color-sense, as Landolt has pointed out, this may be confined to the most central portions of the visual field,—that is, the hysterical patients lose first the power of distinguishing violet and green, while they retain their power of perceiving the other colors. Gradually, it may be, the ability to recognize red and orange disappears, while the power to perceive yellow and blue persists; or the perception of red, instead of that of blue, is the last to disappear. Therefore Pitres has arranged two series giving the order in which the power of recognizing the different colors is lost in hysterical dyschromatopsia: (1) violet, green, blue, yellow, red, and (2) violet, green, red, yellow, blue. In contrast to this, it may be pointed out, as Pitres has done, that in the dyschromatopsia of certain organic conditions,—for example, chronic retrobulbar neuritis, or so-called toxic amblyopia, and tabetic atrophy of the optic nerve,—the perception of red is first lost. While most observers agree in stating that violet or green is the first color to be lost in hysterical cases, Strümpell believes that a definite order of disappearance, such as has been quoted, is entirely too schematic. J. K. Mitchell and myself noted long ago that we were not so apt to find in American patients the dyschromatopsia which has been described, although we did find the inversion of the color field to which full reference has been made, and we also noted that the perception of green was more often disturbed than that of the other colors (violet was not used in our tests), and in this respect the observations coincide with those of Landolt.

Parinaud [26] has noted the following anomalous conditions: A *color scotoma* having all the characteristics of that seen in toxic amblyopia; a central defect with added peripheral contraction resulting in an annular color field in the shape of an intermediary zone between the central circle and the peripheral zone; not only an enlargement of the red field exceeding that for white, but a similar enlargement for other colors (which Pansier maintains is characteristic, page 9), which were either perceived as such or as gray with an intensity greater than that for white. I have recently studied with A. G. Thomson an hysterical boy with a central color scotoma in his field exactly like that which is caused by the abuse of tobacco and alcohol.

Hysterical dyschromatopsia, or disturbance of color-sense manifested by inversion of color lines or loss of certain color perceptions,—for example, violet and green,—may give rise to or be replaced by *hysterical achromatopsia*, a word for which we are indebted to Galezowski. In this condition, first described by Briquet, the patient is unable to distinguish any color, although he recognizes the outlines and shapes of objects. Everything appears, as has been well said, like a sketch in India ink. To the total achromatoptic the colors have a certain luminous intensity, to which brief reference has been made by Obarrio.[27] Investigation of an entirely color-blind intelligent female patient demonstrated that each color matched a certain gray. The patient quickly recognized the equivalent gray tones, indeed, more quickly than a normal individual.

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[26] System of Diseases of the Eye, edited by Norris and Oliver, vol. iv. 1900, p. 736.

[27] Bull. de la Soc. Fr. d'Ophth., vol. xvii. p. 453.

Dyschromatopsia and achromatopsia are apt to be associated with cutaneous and mucous-membrane anæsthesia. Féré maintains that those hysterical patients who have neither narrowing of the visual field nor achromatopsia have normal sensibility of the conjunctiva; those who have achromatopsia and concentric contraction of the visual field have complete conjunctival anæsthesia, and those who have achromatopsia and practically abolished visual fields have not only conjunctival but also corneal anæsthesia. To these observations Gilles de la Tourette ascribes great importance, while Binswanger [28] is unable to confirm the existence of such regular interrelation between the sensory disturbances of the cornea and of the coverings of the eye on the one hand and defects of optical sensation on the other.

(*c*) **Light-Sense and Color-Sense.**—In addition to the concentric contraction of the visual field in hysterical patients, diminution of the light- and color-sense may be present, as has been pointed out by Parinaud, Wolffberg, and particularly by von Frankl-Hochwart and Topolanski. According to the last-named authors, sometimes, although the visual field and light-sense may be normal, whether it be accompanied by disturbances of cutaneous sensibility or not, the power of perception of one or other color—for example, blue—may be defective, and Parinaud states that wherever he has found dyschromatopsia it has been accompanied by modification of the normal white perception. He has also observed patients who, examined with the photometer, could recognize white light only when it was extremely intense, but recognized nothing when this intensity was lowered, and yet who had a luminous impression when the previously unperceived white surface was viewed through a saturated red glass.

The investigation of the color-sense and light-sense in hysterical patients is important, and has not received as much attention as it deserves, because if, as Binswanger points out, there should be an agreement, as there was in von Frankl-Hochwart and Topolanski's examinations, between the restriction of the visual fields and the reduction of the light-sense and color-sense, strong evidence would be present that there was no simulation, which from the perimeter findings alone might be suspected. Binswanger states that Wilbrand was unable with the aid of Förster's photometer to determine anomalies in the light-sense in hysterical amblyopia. The investigations of von Frankl-Hochwart and Topolanski were made with the Chibret instrument.

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[28] Loc. cit., p. 212.

(*d*) **Hyperchromatopsia** has been referred to and the true enlargement of the color fields, to which Pansier ascribes special importance, described. J. K. Mitchell and myself have observed an exaggerated appreciation of colors with corresponding enlargement of the color fields in hysterical hyperæsthesia. This observation has been confirmed by Frankl-Hochwart and Topolanski (Fig. 8). Finally, Binswanger has found in cases of cutaneous hyperæsthesia and hyperalgia marked enlargement of the visual fields for red and green, again confirming the observation which Mitchell and I made many years ago. In hysterical enlargement of the color fields, the blue may exceed all others and pass beyond the white, or there may be inversion of the widened color circles with red as the most peripheral one; rarely this position is taken by green.

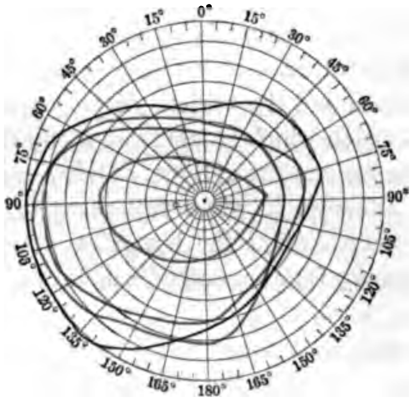

FIG. 8.—Hysterical hyperæsthesia. Patient of Dr. S. Weir Mitchell. Enlargement of color fields and partial reversal of red and blue lines.

Concerning the Nature of Hysterical Visual Field Contraction and Color-Sense Disturbance.—Although hysterics may have extreme contraction of the visual field, they are not inconvenienced by this apparent limitation of their peripheral vision, as is a patient, for example, with pigmentary retinitis. In a word, their orientation is usually perfect. In explanation of this it has been suggested that the peripheral visual impressions, although unperceived, bring about in the muscular apparatus, to use the language of Parinaud, those instinctive and unconscious movements by means of which we find our way through the external world. Groenouw states that hysterical patients with greatly contracted fields find no difficulty in getting about because the periphery of the retina still retains its power of perceiving large and bright objects. Wilbrand explains the phenomenon by assuming that the irritability of the retinal periphery is not lost, and that the insufficiency of the retina is not so great when the patient is moving about as it is during steady fixation. To a certain extent what follows has been discussed under hysterical amaurosis. (Page 619.)

Von Graefe believed that hysterical amblyopia represented an affection of the outer layers of the retina and could, in general terms, be described as a disturbance of the peripheral optical apparatus. Wilbrand has stated that in hysteria certain alterations exist in the visual substance of the rods and cones, and that where there was peripheral contraction of the visual field with retained central vision, these alterations were limited to the peripheral retina, but that where the amblyopia was greater, or true amaurosis appeared, they extended and the entire retina became involved. This theory seemed to be supported by perimetric examinations and the various displacement- and exhaustion-types of the field of vision which result from them, the indication being that the retina is non-resistant to fatigue. (See also, page 683.) Binswanger maintains, although he gives full place to the theories which have been briefly outlined, that the hysterical contracted field is the result of a cortico-sensory fatigue, and of a disturbance of the faculty of concentration.

Pierre Janet [29] regards achromatopsia as consistent with the belief that the retina is sound, but that the visual defect is further back in the perceptive centre. Charcot also maintained that achromatopsia, and therefore dyschromatopsia, are cerebral phenomena. It has been proved that a hysterical achromatoptic viewing red on a white ground will see only a gray image, but after awhile will obtain a consecutive green image. In other words, when the green vibration reaches the perceptive centre, it is not correctly recognized, but nevertheless acts because added to the red vibration it gives the perception of white and produces the consecutive green image.[30] Parinaud, investigating the color contrasts upon hysterical patients, found that the sensation produced by a color that is not perceived can be developed provided the complementary color inducing it is normally seen. The converse of this, however, is not true.

Simulation by the Hysterical Visual Field and Hysterical Amblyopia of Organic Defects.—(*a*) **Hysterical Hemianopsia.**—There is much difference of opinion whether hemianopsia occurs as an ocular stigma of hysteria, and we have statements varying from those of Parinaud, who declares that there is one variety of visual insensibility that hysteria appears to be incapable of producing, and that is hemianopsia, to the assertion of Rosenthal,[31] that in all cases of hysterical amblyopia which he has investigated temporal hemianopsia was present.

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[29] The Mental States of Hysterics, p. 25.

[30] Pierre Janet, loc. cit.

Gowers[32] says that of a large number of cases of hemianopsia which have come under his observation one only may have been of hysterical origin. He further states,[33] "In the hemianæsthesia of hysteria we have evidence of arrested action of all the higher special sense-centres in one hemisphere. In this association we never meet with the hemianopsia that results from disease of the lower centre in the posterior part of the hemisphere. Instead, we have *crossed amblyopia*, as it is termed; peripheral vision is lost in both eyes, central vision persists in a small area in the opposite eye, and in a larger area in the eye of the same side. No trace is found of any relation to the half fields."

Hemianopsia has been reported by Westphal, Galezowski, Svynos, Bonnefoy, M. W. Zimmerman, Henry Lloyd, Dejerine, Koenig, and a number of other observers.[34]

Dejerine and Vialet,[35] referring especially to neurasthenia and traumatic neurosis, describe a special form of hemianopsia which they think constitutes an ocular symptom of the same order as restriction of the visual field, and which is distinguished from the hemianopsia of organic origin by the variability of the limits of the preserved half field, and which may last for years. It is difficult to resist the belief that this represents rather an unusual and irregular contraction of the visual field, with a much greater restriction on one side than on the other, than a true hemianopsia, and, as far as I am able to judge from an examination of the literature, hemianopsia as an *enduring* ocular symptom of hysteria, in the same sense as concentric contraction of the visual field, does not exist.

On the other hand, it must be admitted that as a *temporary* phenomenon it has been observed a good many times by those who are competent to judge, and has appeared either as a bitemporal defect, a homonymous lateral defect, or even, it would seem, as a binasal obscuration. Furthermore, it would seem from the observations of Pierre Janet,[36] who reports a case of transitory hemianopsia which occurred in a woman the subject of grand hysteria, and which he believed was not brought about by suggestion, that it is possible there may always be a period of hemianopsia when the ordinary hysterical amaurosis is gradually recovering. Indeed, such an observation was made by Galezowski and Daguenet long before the publication of Janet's cases. These observers found that hysterical amblyopia gradually recovered, and that between the period of complete recovery and the period of complete amaurosis there was a stage of hemianopsia, and sometimes, instead of this, a transitory central scotoma. A similar phenomenon was present in a patient whose clinical history has been reported by Hirsch in Germany, the defect exhibiting itself in a bitemporal form.

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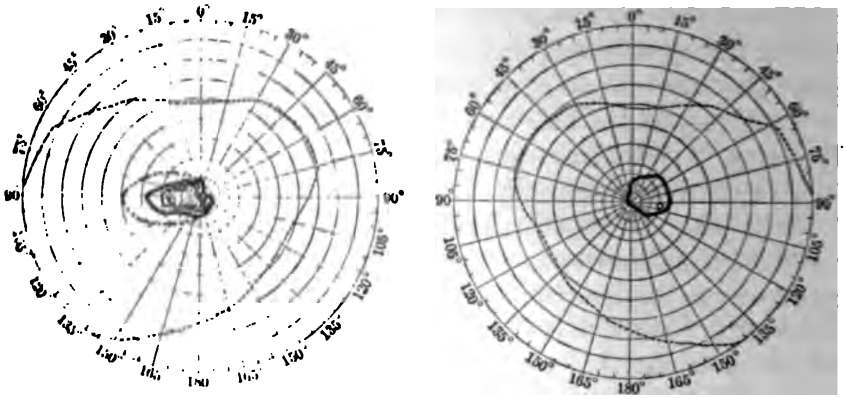
[31] Wiener med. Presse, Nr. 23, 1879, p. 736. In Gilles de la Tourette's book Rosenthal is quoted as having written a letter to Charcot in which he appears to retract the statement just quoted, and says that hysterical patients exhibit only amblyopia and concentric contraction of the visual field, and not hemianopsia.

[32] Diseases of the Nervous System, American edition, 1888, p. 584.

[33] Clinical Lectures, Second Series, Subjective Sensations, 1904, p. 11.

[34] Consult Wilfred Harris, Brain, 1897, p. 308; also Koenig, Rec. d'Oph., 3 Series, 22, 1900, p. 397.

[35] Annales d'Oculistique, 1894, tome cxii.



Left *Right*

FIG. 9. Hysterical field. Patient of Drs. J. K. Mitchell and G. E. de Schweinitz. **Islands of anaesthesia.** Contracted fields and inversion of color lines on left side where the red **exceeds the white field.**

Whether it is true that in place of a homonymous lateral or bitemporal hemianopsia, as Wilfred Harris has described in his Cambridge thesis, binasal hemianopsia may occur I do not know. John K. Mitchell and myself published one case in which, somewhat incautiously, we made use of the expression that the visual fields had the characters of binasal hemianopsia, and since then this case has appeared in literature as an example of hysterical binasal hemianopsia. It should be regarded, I feel sure, not in this light, but as an irregular form of contraction with a greater contraction of the field on the nasal side. (Fig. 9.)

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[36] La Presse Médicale, 1899, p. 243.

According to Gilles de la Tourette, ophthalmic migraine may be accompanied by hemianopsia when it is of hysterical origin but it is always temporary and may be regarded as an exaggeration of the restriction of the visual field. On the other hand, Babinski[37] doubts whether a true relationship between hysteria and an ophthalmic migraine has been established, and believes that the migraine when associated with the hysteria has an independent origin, making the point that between hysteria and hemianopsia there is no marked relationship, while, as he expresses it, the relationship between hemianopsia and ophthalmic migraine is like that of parent to child.

(*b*) **Insular Sclerosis and Hysteria.**—Inasmuch as the symptoms of hysteria may precede the onset of insular sclerosis or occur during its course, this disease may be mistaken for hysteria. Normally, in the visual field between the outer limit of white perception and the outer limit of color perception, colors appear light gray, white, or black, and according to Head[38] the same phenomenon obtains in the field of the hysterical type of disseminated sclerosis, but is not present in the true hysteric field. Referring to the blurred vision, or amblyopia, of insular sclerosis, Buzzard points out that the optic nerve is diseased, or even atrophied, in this disease and normal in hysteria. Other ocular signs suggestive of sclerosis are temporary paresis of an ocular muscle, and especially nystagmus. (See also, Chapter XI, p. 469.)

Kampherstein[39] states that regular functional concentric contraction of the visual field as it occurs in hysterical amblyopia has not occurred in his or in Lübber's investigation of cases of multiple sclerosis. According to Uhthoff,[40] although the occasional association of visual field contraction with insular sclerosis may be explained by a coexisting hysteria, it must be remembered that in the former this condition may obtain, even in the absence of ophthalmoscopic change, and be none the less occasioned by lesion of the opticus. Therefore the diagnosis of pure functional disturbance of the visual field under these conditions is difficult.

(*c*) **Syringomyelia and Hysteria.**—Concentric contraction of the visual field for all colors has been reported in syringomyelia by Dejerine and

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[37] Archiv de Neurologie, xx. 1890, p. 305.

[38] Quoted by Buzzard, British Medical Journal, Oct. 7, 1893.

[39] Archiv f. Augenheilk., Bd. xlix. Erstes Heft, 1903, p. 41.

[40] Graefe-Saemische, Handbuch der gesammten Augenheilkunde, Lief. 68–71, 1904, p. 352.

Tuilant, but Gilles de la Tourette agrees with Brianceau, whom he quotes, that in the majority of cases of this affection the visual field is normal, and that when it is contracted it is necessary to search elsewhere than in the disease itself for this phenomenon. Hysteria, which may accompany the affection, is accordingly given in this respect the etiological rôle, in the absence of ophthalmoscopic lesions. (Chapter XV.)

(*d*) **Exophthalmic Goitre and Hysteria.**—Concentric contraction of the visual field has been observed in exophthalmic goitre, but in the experience of Gilles de la Tourette, under these circumstances hysteria has always been associated with the malady. Personally, I have examined a great many cases of exophthalmic goitre, and in the absence of ocular lesions have not found notable alterations in the visual field, certainly nothing similar to those which have been described as more or less characteristic of hysteria. (See Chapter XIX.)

(*e*) **Epilepsy and Hysteria.**—It is well known that there may be contraction of the visual field after epileptic attacks, and not only a mere contraction of the visual field but an amblyopia, and therefore in these respects an analogy between this disease and hysteria is recognized. Furthermore, repeated epileptic attacks might lead to an apparent permanent contraction of the visual field or, to speak more accurately, a temporary amblyopia which would rapidly subside after one attack might persist longer should the attacks be many times repeated. Parinaud,[41] after quoting the observations of Oppenheim and Thomsen, that contraction of the visual field is frequently seen in epilepsy, records Hitier's examinations in Charcot's clinic, where he found in 87 cases of epilepsy a permanent restriction of the visual field in only three. Therefore, as there is an occasional more than temporary amblyopia of this character, it is necessary that the differential diagnosis of epilepsy and hysteria from the visual stand-point should not rest alone upon the phenomena connected with the field of vision. The other ocular symptoms of hysteria should serve to establish the diagnosis. Finally, there is no reason why there should not be an association of epilepsy with hysteria, an association which has not infrequently been observed. (Chap. XVII.)

(*f*) **Retrobulbar Neuritis and Hysteria.**—It will at once be apparent that sudden amaurosis of hysteria and the rapid loss of vision which accompanies retrobulbar neuritis on superficial examination bear a marked similarity, and there is no doubt that the one has occasionally been mistaken for the other. Naturally, the reaction of the pupil gives important information, and if there is blindness with preservation of pupillary contraction to light stimulus, retrobulbar neuritis may be excluded. Sometimes, however, as we shall later see, the pupil does not react to light, temporarily at least, in hysterical cases, and therefore on this point alone a differential diagnosis could not be established, and the other symptoms which have been described in connection with hysterical amaurosis would almost certainly lead to a proper diagnosis,—for example, the disappearance of monocular blindness in binocular fixation, anæsthesia of the conjunctiva, and contraction of the visual field of the opposite eye and inversion of the color lines. Symptoms of hysteria not associated with the eye would add most important diagnostic information.

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[41] System of Diseases of the Eye, edited by Norris and Oliver, vol. iv, p. 763.

The method in which the blindness occurs in retrobulbar neuritis is usually characteristic, inasmuch as the obscuration of vision starts in the centre of the field and progresses in from one to eight days to complete or nearly complete blindness; furthermore, there is the comparatively rapid replacement of negative ophthalmoscopic signs with alterations in the fundus oculi, and pain is evident on movement of the eyeball and when it is pressed backward into the orbit.

Some authors—for example, Bregmann[42]—suggest that just as retrobulbar neuritis may depend upon a primary disturbance of metabolism, so also may hysteria, the unknown toxin working on the one hand on the optic nerve and on the other on the brain, and think it also possible that the optic nerve disturbances may depend upon vasomotor changes in the opticus. Hysterical œdema is well known, and therefore it is possible that an œdema might arise in the optic nerve which would bring about a condition of elevated pressure in the optic-fibres, especially in the neighborhood of the optic foramen.

Not only has restriction of the visual field similar to that seen in hysteria been found in the diseases which have been quoted in the preceding paragraphs, but in many other organic conditions,—for example, brain tumor, lesions of the posterior portion of the internal capsule (Charcot), tabes dorsalis, Friedreich's ataxia, and, finally, in certain toxæmias, notably those produced by lead, alcohol, mercury, and bisulphide of carbon. Indeed, for a long time, particularly in France, certain toxic agents have been regarded as capable of originating hysteria, and the whole subject has been well elaborated by Goinon.[43] Sometimes not only is there contraction of the visual field, but reversal of the color lines, as in Nieden's case of nitrobenzol poisoning.[44]

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[42] Zeitschrift f. Augenheilk., vol. v. 1901, p. 391.

[43] Les Agents provocateurs de l'Hystérie, Paris, Thèse, 1889.

[44] Consult The Toxic Amblyopias, by G. E. de Schweinitz, pp. 130, 231.

III. DEFECTIVE AND PAINFUL VISUAL SENSATION, OR HYSTERICAL ASTHENOPIA.

Hysterical Asthenopia.—Thus far the visual phenomena of hysteria have been representatives of various types of anæsthesia of the visual sense, or, in other words, a diminished or abolished sensibility to visual impressions. In contrast, a number of patients of the hysterical type are afflicted with markedly painful visual sensations, notably dread of light, lachrymation, blepharospasm, neuralgic pains which appear in the neighborhood of the ocular globe and radiate to the temporal and zygomatic regions, and imperfect eye endurance. To this set of symptoms the term *hyperæsthesia of the retina* has sometimes been applied. They represent the condition which Förster described as *hysterical kopiopia*, Donders as *painful accommodation*, Nagel as *hyperæsthesia of the ciliary muscle*, and Schenkl as *hysterical ocular pain*, but which, in general terms, may be classified under the name attached to the beginning of this paragraph,—namely, painful visual sensations, or hysterical asthenopia.

Most frequently encountered in women, and not rarely among children, it is probable that the disorders of accommodation and of convergence which will be described in subsequent pages are always present, and are responsible for the photophobia and other symptoms so commonly supposed to indicate hyperæsthesia and irritation of the retina. Any effort of vision, particularly at close work, is quickly followed by ocular pain, brow neuralgia, and diffuse headache, often with micropsia and obscuration of vision, and quickly by fatigue. The intolerance of light, whether it be diffuse daylight or artificial light, is relieved by tinted glasses, to which the patients naturally turn.

Schwarz believes that to hysterical weakness of sight with reduction of visual acuity, differentiated from pronounced hysterical amblyopia, should be accredited all the cases which were formerly called "anæsthesia and hyperæsthesia of the retina," "amblyopia of the visual field," and "nervous asthenopia," but includes with the symptomatology concentric contraction of the visual field, inversion of the color lines, disturbance of the light- and color-sense, and sometimes central scotoma. It would seem, however, that a large majority of these cases present only the symptoms before detailed,—namely, painful visual sensations, lack of eye endurance, photophobia, chromatophobia, etc. We have in these cases, according to Binswanger, to deal with hyperalgesia manifesting itself through light impressions, and he believes that these hyperalgesic conditions form part of a general hyperalgesia, most pronounced in certain forms of hystero-neurasthenia. I shall, when discussing neurasthenia, refer to this subject again, as the best types of nervous asthenopia of this particular variety are seen in the neurasthenic rather than the hysteric patient. (See Page 672.)

Hysterical Ophthalmic Migraine.—At this point reference should be made to the association of ophthalmic migraine and hysteria, constituting the so-called "hysterical ophthalmic migraine" described by a number of authors.[45] The well-known symptoms of ophthalmic migraine—paroxysmal headache, hemicranial in type and sometimes proceeding to the opposite side, ushered in by visual phenomena, the most important of which are various types of expanding and scintillating scotomas and hemianopsia, the attack terminating in nausea and vomiting—occur in hysteria. According to Pansier, the migraine may constitute the aura of the hysterical attack, or be contemporaneous with it, or be accompanied with symptoms which are undeniably hysterical, or, finally, be brought into existence by pressure on the migrainous points analogous to the hysterogenetic zone, or by suggestion. Whether hemianopsia may accompany hysterical ophthalmic migraine has been especially studied by Babinski and Parinaud, and Gilles de la Tourette, as before stated (compare page 637), concludes that if it is present it exists only as a temporary phenomenon, or, more accuratèly, seems to be a temporary exaggeration of the concentric restriction of the visual field. (Compare Chapter XVII.)

IV. ANOMALIES OF ACCOMMODATION, OR HYSTERICAL CYCLOSPASM AND CYCLOPLEGIA.

It is generally conceded that the most frequent ocular manifestations of hysteria are those concerned with anomalies of accommodation, particularly in the form of

(1) **Hysterical Accommodation-Spasm, or Cyclospasm.**—This condition may be associated with amblyopia or occur independently of it, and is seen with particular frequency in young subjects. It may be partial or total, unilateral or bilateral, and has been particularly studied by Parinaud.[46] According to this investigator, hysterical spasm of accommodation is similar to that produced by eserin, and has as its chief characteristic a displacement towards each other of the *punctum*

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[45] Consult Charcot, Leçon de Mardi, 1887–1888, p. 101; Babinski, Archives de Neurologie, November, 1890; and Fink, Des Rapports de la Migraine ophthalmique avec l'Hystérie, Thèse, Paris, 1891.

[46] Annales d'Oculistique, tome lxxix. 1878, p. 218; ibid., tome xcvi. 1886, p. 47; see also System of Diseases of the Eye, edited by Norris and Oliver, vol. iv. p. 747, "The Ocular Manifestations of Hysteria," by Parinaud.

proximum and the *punctum remotum*,—that is to say, there is a diminution of accommodation at both ends of the line. It will be observed that such phenomena are precisely opposite to those which occur in paralysis of accommodation, where the punctum proximum approaches the punctum remotum, and when it is coincident with it there is complete abeyance of the function of accommodation. In the hysterical patient, as long as p and r do not coincide, there is some range of accommodation; when by virtue of the continuance of the displacement of r towards p a point is reached where fusion takes place, accommodation ceases, and the patient can see only at a fixed point. (See Page 37.)

From this description of Parinaud's it will be seen that the recession of p brings about a state of affairs resembling paresis of accommodation, while the advance of r creates an apparent myopia. The former condition would be neutralized by a convex glass, the latter by a concave glass. In illustration a recent case under my care may be briefly quoted: A young hysterical girl, whose refraction was a hypermetropia of 1 D., during an attack of spasm of the accommodation required —1 D. to raise her vision to the normal standard, while at the same time her amplitude of accommodation was only 3 D.,—that is, her near-point for fine print was 33 centimetres, which was brought back with a suitable convex glass to the normal point,—namely, 12 centimetres.

As Parinaud points out, the point of fusion may be more or less remote from p or from r without losing the character of spasm, and the distance of the fusion point from p and r must necessarily be influenced by the refractive condition of the eye,—that is, whether the eye is myopic, hyperopic, or presbyopic. It will therefore be seen that, according to circumstances,—that is, according to whether there has been coincidence of p and r,—certain symptoms must obtain, which, to repeat, are these: An apparent weakness of accommodation at the near-point, a defect of distant vision or a *spasmodic myopia*, and as a result of the contracture of the ciliary muscle in a manner presently to be explained, monocular diplopia, sometimes polyopia, and not infrequently micropsia and megalopsia.

The *degree* of *apparent myopia* from cramp of accommodation in hysterical patients varies. I have seen in an hysterical woman with hypermetropic refraction as follows, +.75s+.50c axis 90, a cramp represented by concave 7 D., which restored the visual acuity practically to the normal standard. The most remarkable case of this character with which I am acquainted is that reported by Loeser.[47] In an hysterical boy without atropin a vision of 5/10 was obtained with a —24 D. in one eye and a —22 D. in the other. After atropin the refraction of the right eye was + 2c axis 10 and of the left eye + 1.50c axis 25.

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[47] Centralbl. f. prakt. Augenheilk., xxvi. 1902, p. 170.

The other symptom to which reference has been made, namely, *monocular diplopia* and *polyopia,* is not always manifest unless sought for by the methods of functional testing,—that is, the patient does not necessarily describe its presence. Usually, when the test-object,—for example, a small point of light,—is held at a certain distance from the eye, it is seen singly, but at points nearer or further from that at which single vision is obtained the diplopia appears. According to Parinaud, the second image usually separates from the first on the temporal side, but a vertical displacement, or one obliquely situated from the test-object, is also seen. Occasionally *polyopia,* or, more accurately, *triplopia,* is evident,—that is, a third indistinct image is observed upon the opposite side. According to Parinaud, when it appears beyond the point of single and distinct vision it can be made to disappear by the use of a concave glass, but when it is seen on the proximal side of the point of single vision, it disappears if viewed through a convex glass.

A partial contraction of the muscles of accommodation has been described by Borel, Galezowski, and Pansier. This irregular spasm of the ciliary muscle creates more or less astigmatism, which disappears with the restoration of the patient's general health.

Schwarz[48] insists, in his description of these phenomena, on the difference of their behavior in binocular and monocular vision. According to him, in binocular vision hysterical accommodation disturbances are rare. In high grades of accommodation-spasm there is usually an associated convergence-cramp. The cyclospasm appears only during fixation, and the object regarded, at first plainly seen, becomes blurred and double vision appears. Should this double vision continue, even if one eye is covered, the condition is one of monocular diplopia, often associated with micropsia. If, on the other hand, the double vision disappears when either of the eyes is covered, binocular diplopia exists, and there is, if the diplopia is homonymous, hysterical convergence- and accommodation-spasm, provided paresis of the abducens is excluded. If the double vision is crossed, then there is weakness or paresis of convergence, provided organic disease is absent.

Another symptom connected with hysterical spasm of accommodation is an inability properly to estimate the size of a test-object which is fixed.

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[48] Die Bedeutung der Augenstörungen für die Diagnose der Hirn- und Rückenmarks-Krankheiten, Berlin, 1898, p. 80.

For example, the test-object appears to grow larger when it is made to approach the eye, and appears to grow smaller when it is carried to a distant point, corresponding in this respect to the size of the retinal images. To this symptom Parinaud has given the name *micro-megalopsia*, and he considers it an almost constant one in patients suffering from hysterical amblyopia and spasm of accommodation.

(2) **Hysterical Paralysis of Accommodation, or Cycloplegia.**—Although paralysis of the ciliary muscle, which has been observed to exist both in a partial and a complete condition, is uncommon as compared with spasm of accommodation, it is not unusual to see in young hysterics, and especially in hysterical children, mild degrees of accommodative weakness, so that the patient is unable to read fine print at the distance normal to the age and to the condition of refraction, and is able to do so only with the aid of convex glasses. As has already been pointed out, this does not necessarily mean that there is a paresis of the ciliary muscle, because we have seen that while there may be a recession of the near-point there is usually an advance of the far-point, and spasm is therefore not excluded, the condition being one of contracture of accommodation.

On the other hand, in common with many other observers, I have a number of times, particularly in mild types of hysterical manifestations, often those following injury in young people, observed undoubted paresis of accommodation comparable in all particulars to an imperfect power of the ciliary muscle, such as might be produced if a cycloplegic had not exercised its full power. Wilbrand and Saenger have recorded instances of paresis of accommodation with transitory diplopia in nervous children, and have demonstrated that these temporary conditions of weakness of accommodation may pass on to paresis of the ciliary muscle. They are well known as part of the symptomatology of hysterical asthenopia. (See Page 640.)

According to Schwarz,[49] hysterical accommodation-paralysis is usually bilateral, and generally associated with palsy of convergence. If this is present and there exists at the same time no divergent strabismus at the distant point, the hysterical nature of the disturbance is probable. The same author insists, if convergence is normal, that the binocular range of accommodation should be tested, after adjustment of any difference in refraction of the two eyes. Should, under these circumstances, the binocular accommodation range be essentially greater than the monocular, the hysterical nature of the paralysis may be assumed, because with convergence in binocular vision the apparently paralyzed accommodation comes into action. If the accommodation in binocular vision, in spite of good convergence, remains as much in abeyance as in monocular vision, organic paresis is probably present. This opinion is disputed by Blok,[60] who reports double hysterical spastic mydriasis accompanied by bilateral paralysis of accommodation. So-called typical cases of paralysis of accommodation, with diplopia, have been recorded by Nonne and Beselin; they are especially apt to occur if the refraction is hyperopic.

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[49] Loc. cit., p. 82.

Schwarz's observation of the disappearance of unilateral hysterical accommodation-paralysis in binocular vision corresponds to the subsidence of monocular amaurosis in binocular fixation. In other words, the unilateral paralysis of accommodation is apparent only, and Schwarz believes that in these cases it has not been proven that the palsy persisted during binocular vision. Therefore he doubts the occurrence of such a pure unilateral hysterical cycloplegia.

Although the pupils are usually normal in cases of hysterical cycloplegia, this is not necessarily the case, and mydriasis has been reported, —for example, by Donoth, Nonne and Beselin, and Bloq, the last-named reporter, as stated above, having recorded a case of mydriasis and accommodation-paralysis, at first alternating from the right to the left eye, but later becoming stationary in both eyes, and associated with convergent strabismus. The action of eserin was only slightly manifested.

Concerning the Pathogenesis of Hysterical Accommodation-Disturbances.—As has already been sufficiently stated and largely proved by Parinaud, the most usual basis of the phenomena under consideration is a *spasm of the ciliary muscle*, and this spasm, if complete, wipes out the amplitude of accommodation because the eye is adapted for a fixed point at which there is single vision. On either side of this fixed point diplopia occurs, and the cause of this phenomenon is referred by Parinaud to structural defects in the crystalline lens. He writes as follows: "The disposition of the anterior portion of the lens in three segments favors its changes in curvature, but at the same time, owing to their separation from one another by layers of amorphous material, each section possesses a focal point of its own, capable of producing images distinct from that yielded by the lens as a whole. The condition of spasm by increasing the convexity of the anterior surface of the lens serves to emphasize the diplopia arising from the structural defects described." He believes that in those cases in which anomalies of the central nervous system have been brought forward to explain multiple images, the condition of the refraction has not been perfectly examined. On the other hand, Pierre Janet, while admitting Parinaud's explanations, is unwilling to deny the probable psychic origin of some of these phenomena. The probable explanation of micropsia and megalopsia has been described, and the interesting waking up of accommodative effort (which was in abeyance in monocular vision) during binocular fixation has been recorded.

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[60] Annales d'Oculistique, cxix. 1898, p. 200.

V. ANOMALIES OF THE IRIS MOVEMENTS, OR HYSTERICAL PUPIL PHENOMENA.

Pupil disturbances of hysterical origin have occasioned much dispute, and Schwarz's statement that observations concerning anomalies of the iris movements of hysterical origin are often inexact is undoubtedly true, as is also the remark of Parinaud that pupillary anomalies occur rarely as permanent manifestations of ocular hysteria. The following conditions have been reported to be present in hysteria:

(1) Contraction of the pupil,—that is, either spasmodic or paralytic myosis.

(2) Dilatation of the pupil,—that is, either spasmodic or paralytic mydriasis, the dilated pupil in one class of cases responding to light impulses, even when light perception is denied, and in another class of cases failing to contract under the influence of light impulse.

(3) Interchange of mydriasis and myosis.

(4) Unilateral mydriasis in one class of cases unassociated with paresis of accommodation, in another class of cases associated with paresis of accommodation.

(5) Dilatation of the pupil with preserved but sluggish reaction to light and paralysis of accommodation.

Contraction of the pupil, or hysterical *spasmodic myosis*, as an isolated symptom, if it occurs at all in hysteria, must be exceedingly rare, although myotic pupils, according to Westphal,[51] may appear independently of hysterical seizures, or at the conclusion of them, or sometimes during trivial attacks. In so-called hysterical pseudomeningitis, myosis has been described (Boissard, quoted by Gilles de la Tourette), but whether the myosis was spastic or paralytic is a matter of doubt. Hysterical rigidity of the pupil has been observed by Spiller. Dilatation of the pupil following myotic contracture has been reported, and Gilles de la Tourette considers this preliminary contraction of the pupil important. He doubts whether there is a true paralysis of the sphincter of the iris which causes the subsequent mydriasis, and attributes the dilatation to a preponderating effect of the dilator after the contracted iris has relaxed. The presence of pupil contraction in association with accommodation and convergence cramp, according to Schwarz, is undoubtedly often present, and Galezowski has noticed myosis as an accompaniment of spasmodic myopia, as it has already been described in a previous section, an observation which Parinaud has not been able to confirm. In general terms, the myotic pupils, which should be ascribed to a spastic condition of the sphincter of the iris, are brought about by psychic disturbances, and as already noted, may appear independently of the attacks, or at the conclusion of them; when the psychic disturbance is removed the pupil may dilate.

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[51] Berlin. klin. Wochenschr., xxxiv., 1897, p. 1024.

Spasmodic mydriasis in connection with hysteria has been described frequently. The literature has been reviewed by Aurand and Frenkel,[52] Nonne and Beselin,[53] Binswanger,[54] and Pichler,[55] and from a study of this literature Binswanger asserts that although hysterical mydriasis is rare, it has a true and autonomous existence. Clinically it is necessary to distinguish between a spasmodic and a paralytic form, either of which may be independent, but Binswanger thinks it must be stated that a patient may have at the same time spasmodic and paralytic mydriasis. We must further grant that in one class of cases of mydriasis, even if associated with amaurosis, there is response of the pupil to light impulse, while in another class of cases there is failure of the iris to contract under the influence of light. Again, the dilatation of the pupil may or may not be associated with paralysis of accommodation. In many of the cases of mydriasis there is amaurosis, and the mydriasis disappears when sight is restored, but Aurand and Frenkel think that it may occur quite independently of amblyopia, or even a greater depreciation of vision. Thus, Parinaud and Richer report a case of hysteria with paralysis of accommodation and mydriasis, Harlan one of mydriasis, accommodation-paralysis, and blepharospasm, while, according to De Wecker, in hysteria the mydriasis may be associated with cycloplegia or exist without it. Touching the question of whether the mydriasis is a paralytic one or not, Schwarz maintains that although it is often so stated, such statement is not necessarily based upon accurate observation. Even when the pupil, widely dilated, does not react to light, it is not necessary that a paralytic mydriasis should be assumed. A pure paralysis of the iris movement not brought into existence by a medicament can be stated to exist only if the pupil promptly responds to the action of pilocarpin or eserin. Even the presence of hysterical cycloplegia does not prove, according to him, that the mydriasis is paralytic, for, on the one hand, one may have paralysis of accommodation without pupil paralysis, as, for example, in diphtheria, and, on the other hand, as has already been shown, the so-called hysterical accommodation-paralysis is not a paralysis in the true sense of the word. The cases of paralytic mydriasis reported by Roeder, Donoth, and Nonne and Beselin have not been accepted by all authors. It has been suggested, for example, that atropin might have been used without knowledge, and although in Nonne and Beselin's case eserin failed to contract the pupil, and a central paralysis and a spastic mydriasis were undoubtedly excluded, and although the patient was in a certain sense watched in so far as the use of atropin is concerned, Schwarz is evidently unwilling to accept the paralytic nature of the mydriasis, which these authors, however, do not hesitate to maintain, describing it as an undoubted paresis of the sphincter of the iris in a case of typical grand hysteria. They record a second case which they describe as *hysterical ophthalmoplegia interna*. Liebrecht, on the other hand, remarks that he has never seen an undoubted case of hysterical internal ophthalmoplegia.

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[52] Revue de Médecine, xvi. 1896.

[53] Festschrift zur Feier des achtzigjährigen Stiftungsfestes des Aerztlichen Vereins zu Hamburg, Leipzig, 1896.

[54] Loc. cit., p. 628.

[55] Zeitschr. f. Augenheilk.

As has already been stated, mydriasis in the absence of paralysis of accommodation is due to a spasm of the dilator muscle. To explain the paralytic mydriasis, Nonne and Beselin, as well as Donoth, assume an abeyance of the cortical centre governing the functions of the sphincter of the iris and the muscle of accommodation, and in this respect establish an analogy between the condition described in so far as etiological considerations are concerned, and hysterical amblyopia, amaurosis, dyschromatopsia, and concentric visual field contraction. Binswanger, although he does not doubt the occurrence of pupillary rigidity or pupillary sluggishness as an hysterical symptom, warns against its being mistaken for reflex light rigidity in the narrow sense of the term, which belongs to the domain of the organic diseases of the brain and spinal cord, pupillary rigidity in hysteria being associated with myosis and spasm of accommodation and convergence. Deutschmann, Wilbrand, and other observers also point out the necessity of making a distinction between a true reflex pupillary rigidity and mydriatic pupils which do not react either to light or accommodation. In response to such objection Nonne and Beselin point out that they do not describe reflex pupillary rigidity, but only mydriatic pupils which fail to respond to light and convergence, and again maintain that the condition is due to functional injury of the cortical centres for the innervation of the sphincter or of the dilator.[56]

Pupil dilatation may exist for a short time, for a comparatively long period, and in rare instances,—for example, in Harlan's and in Nonne and Beselin's cases,—for several months or even years. The pupil may be made to contract by suggestion, hypnotism, and by causing the patient, as in Pichler's case, to read through a plane glass.

Thus far we have spoken particularly of bilateral dilatation of the pupil. There may be, however, a *unilateral mydriasis*, sometimes associated with paresis of accommodation and sometimes without such association, and there may be dilatation of the pupil with preserved but sluggish reaction to light and so-called hysterical paralysis of accommodation. Gilles de la Tourette describes a hypothetical unilateral mydriasis in total amaurosis of one or both eyes as the result of the loss of the light reflexes, which under normal conditions influence the pupillary reaction, but Binswanger, commenting on this case, thinks that there are some cases in literature belonging to this category,—for example, those reported by Mendel. H. A. Hare and myself have recorded[57] complete amaurosis with mydriasis of the right side, the dilated pupil reacting sluggishly to light impulses and moderately to eserin, with marked myosis of the left side and moderate amblyopia, in a hysterical woman, which had existed for many years, the use of drugs being absolutely excluded by keeping her under the observation of trained nurses for weeks at a time in a hospital room.

In addition to immobility of the pupil there is sometimes an *interchange of myosis and mydriasis*, and Lagovides reports a case of hysterical alternating and intermittent mydriasis, while Blok has observed a case of mydriasis and accommodation-paralysis at first alternating from the right to the left side, but later stationary in both eyes, associated with strabismus convergens.

Before dismissing this subject the condition of the pupils during hysterical attacks remains to be described. We have already noted that pupil immobility may occur during an attack, at the end of an attack, or without evident association with an attack, although permanent pupil immobility has not been observed.

Strümpel, Oppenheim, and Möbius at one time maintained that a differential diagnosis between epilepsy and hysteria could be made by observing the pupils, which in the former were wide and reactionless, while in the latter affection their reaction was preserved. Karplus[58] first, in 1896, observed pupil-immobility during hysterical attacks, and a year later Westphal established the accuracy of Karplus's contention that the state of the pupils was not sufficient to make a differential diagnosis between epilepsy and hysteria. Karplus still later accurately investigated a series of cases of spontaneous and induced hysterical attacks, during which the pupils were dilated and immobile. He considers reflex pupil immobility a frequent phenomenon in paroxysmal hysteria, and believes that it depends upon a cortical origin, the pupil-dilatation and pupil-contraction being represented in the cortex, and that a tonic cramp of the muscular structure of the iris can occur, and therefore light reaction not take place, exactly as a tonic cramp of the legs in hysteria may be responsible for the elimination of the patellar reflex.

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[56] For further discussion of this subject and the views of neurologists and ophthalmologists, see Binswanger, loc. cit., p. 637.

[57] American Medicine, April 21, 1901.

Finally, we may have in hysteria a *paradoxic pupil reaction* which has been reported by Westphal and by Lepine. The last-named author's patient showed dilatation of the pupil when light fell upon the exposed eyes, this dilatation remaining for a few seconds. The transitory and inconsistent changes in the pupil, to use Binswanger's words, which are seen in so many hysterical patients, should be regarded chiefly as an expression of a pathological perversion by virtue of which psychic processes are more easily transmitted to the movement of the pupil than under normal conditions. Thus it is that we sometimes have a wide but quickly reacting pupil,—that is, one which dilates rapidly under the influence of shock, excitement, exertion, etc.,—and also, as has often been observed, the increase of the natural *hippus* of the pupil. I have seen in one hysterical patient of Dr. Weir Mitchell the most marked example of exaggerated hippus that has come under my observation, except as I have seen it in one or two cases of insanity. Von Kétley[59] describes a *myasthenic action* of the pupils in hysteria. (Compare Chapter VI.)

VI. ANOMALIES OF THE EYELID AND EYE-MUSCLE MOVEMENTS, OR HYSTERICAL SPASMS, CONTRACTURES, PSEUDOPALSIES, AND PALSIES OF THE PALPEBRAL AND BULBAR MUSCULATURE.

1. **Blepharospasm.**—This condition appears in a clonic, tonic, and so-called pseudoparalytic form.

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[58] Lehrbuch f. Psychiatrie, xvii. 1898, pp. 1–53.

[59] Wien. med. Wochenschr., Nr. 33, 1901, p. 1559.

(*a*) **Clonic blepharospasm,** common enough in hysteria, is not necessarily an ocular stigma of this affection. It is characterized by a vibratory and continual blinking of the lids during the attack. Usually bilateral, clonic spasm of one orbicularis appearing in the form of fibrillary contraction has been observed. Frequently part of the symptomatology of an hysterical seizure, it may arise independently of such association. In one form of the affection described by Bernutz as the *attenuated variety,* there is a constant vibration or trembling of the closed or half-closed lids, which does not cease even during sleep.

(*b*) **Tonic Blepharospasm.**—This is characterized by a convulsive closure of the eye, and, unlike the clonic variety of this affection, it is frequently, if not usually, unilateral, or at least is more marked upon one side than the other, and the spasm may involve the neighboring muscles of the face and neck. The skin of the lid is forcibly folded and the palpebral borders are vigorously applied one against the other. If an effort is made to raise the lid,—for example, by means of the finger,—a definite resistance is demonstrable; and if the patient under command tries to open the eye, the only response is a trembling movement without separation, or without distinct separation, of the ciliary margins which have been in contact with each other. Along with this effort there may be a distortion of the face and contraction of other muscles,—for example, those opening the mouth. Quite commonly blepharospasm is associated with intense photophobia, convulsive displacement of the eyes in different directions, dyschromatopsia, spasm of accommodation, and amblyopia. Indeed, it was originally believed by Hocken that blepharospasm always accompanied amaurosis, although we now know that such is not the case.

Two clinical varieties have been described, the *painful* and the *non-painful.* The former, usually bilateral, may be accompanied by the visual troubles before named, especially lachrymation and photophobia, while the latter, or the non-painful form, is frequently unilateral. Gilles de la Tourette has described in painless blepharospasm an anæsthetic zone around the eye extending for a distance of two or more centimetres above and below the orbit. The conjunctiva is insensitive, although the cornea may still retain its sensibility, except at the internal portion. In painful blepharospasm with photophobia the zone of anæsthesia is replaced by one of hyperæsthesia, which may extend to the cornea. Richer and Gilles de la Tourette have noted that compression of the supraorbital region may cause momentary disappearance of the blepharospasm, while pressure upon other areas in the neighborhood of the eye may cause an exacerbation or a relapse after quietude. Almost complete cessation of the blepharospasm of one of my patients could be induced by hard pressure in the neighborhood of the supraorbital foramen. Furthermore, the blepharospasm—as, for example, in Zehender's patient—may be exaggerated or called into existence by pressure on the first cervical vertebra or on the sternal region or, as in Seeligmüller's case, on the lower jaw in the neighborhood of the molar teeth.

The condition may develop without any external cause in conjunction with or after an hysterical crisis, or very frequently in the ordinary reflex manner, after a conjunctivitis, a corneal affection, or a sudden flash of light. Sometimes the blepharospasm arises suddenly and disappears with suddenness or, the blepharospasm being complete, convulsive movements of the lids in the form of exacerbations occur spontaneously, lasting from a few minutes to several hours. Contracture of the orbicularis does not always disappear suddenly; and Charcot and Gilles de la Tourette have published cases in which it has lasted for several years. Again, as in Harlan's patient, there may be an *intermittent* violent blepharospasm of one eye, with contraction of the pupil, spasm of accommodation, and narrowing of the visual field.

Binswanger regards blepharospasm as a concomitant of the general pathological tendency in hysteria to the formation of contractures. According to Gilles de la Tourette, its origin should be considered central. Other authors believe that contraction of the orbicularis, at least certain varieties of it, are due to hyperesthesia of the retina, which causes a reflex closing of the lid to protect the eye from the excessive light.

(c) **Pseudoparalytic Ptosis.**—In spite of this name, which we owe to Parinaud, as we do also in large measure the differentiation of the affection from a true ptosis, this condition is not the result of levator weakness, but depends upon a cramp or spasm of the palpebral portion of the orbicularis. Therefore it would seem that the other term *spastic ptosis* is the more appropriate one.

According to Parinaud, pseudoparalytic ptosis resembles palsy of the levator because the upper lid droops without wrinkling the skin of the lid, and if the patient attempts to open the eye there is a contraction of the occipitofrontalis similar to that which one sees in real paralytic ptosis. The lid raised with the finger, however, when released falls more rapidly than in paralytic ptosis, and the patient may, if his attention is directed elsewhere, voluntarily elevate the lid, or as dusk comes on, because photophobia and amblyopia are frequently accompanying symptoms, perform the same act. According to Charcot and Landolt, in true paralysis of the levator the eyebrow is elevated, inasmuch as the patient endeavors to lift the eyelid by contracting the occipitofrontalis, while in pseudoptosis, which is really due to a partial spasm of the orbicularis, the eyebrow follows the contraction of this muscle and descends below the normal level, and there is obliteration of the wrinkles on the corresponding side of the forehead (Fig. 10). This important observation is of particular interest because we may have, as Charcot has shown, a simulation by hysteria of the syndrome of Weber,—that is to say, a ptosis on the side opposite to hemiplegia. It would seem, however, that this so-called *eyebrow-sign* is not a constant one, as Wilbrand and Saenger have shown, and that the other indications referable to blepharospasm must be sought out and examined. Sometimes the depressed eyebrow is straighter than the one on the opposite side; the overhanging folds of skin of the upper lid are converted into a series of parallel or concentric folds, the free border of the lid is curved, the occipitofrontalis muscle comparatively smooth, and the palpebral fissure not entirely closed. Finally, there may be some resistance to the finger in the effort to raise the lid, and the pseudoptosis does not disappear when there is over-extension of the head. Binswanger, who elaborates these differential diagnostic points, thinks they are important to support the diagnosis of pseudoptosis in doubtful cases, and calls attention to the fact that a sharp distinction between spastic ptosis and fully developed blepharospasm is often difficult, especially as there are transitory forms

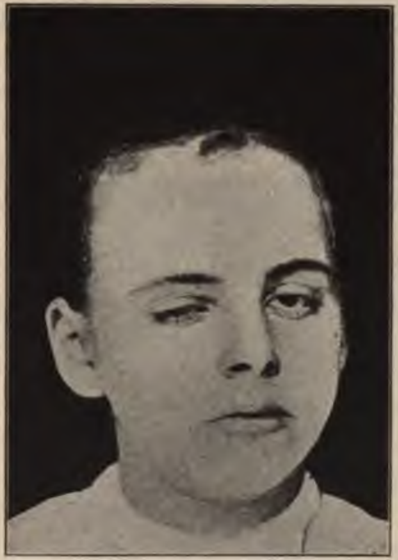

FIG. 10.—Hysterical spastic ptosis. (After Wilbrand and Saenger.)

—as, for example, in Zehender's case, in which blepharospasm terminated in pseudoptosis.

Although spastic ptosis is usually unilateral, bilateral varieties of the affection have been reported, and the condition may be accompanied by hemispasms elsewhere,—for example, glossolabial hemispasm, as in a case of Gilles de la Tourette's. Binswanger points out that hyperæsthesia of the retina may be a precursor of pseudoptosis, and, quoting Bardol's case, insists that sometimes psychic causes alone are operative. He agrees with Wilbrand and Saenger that a palpebral spasm brought about, as in the case recorded, by a flash of lightning, represents a pathological persistence of the voluntary closing of the lids to shut off the sight of the storm, and demands that spastic ptosis in hysteropathic individuals with overexcitability in the sensory sphere should be regarded as a pathological exaggeration of physiological, cortical, and intracortical reflex.

2. **Hysterical Paralysis of the Orbicularis.**—According to Gilles de la Tourette, the title just given is a misnomer, as palsy of the orbicularis has not been proved to occur in hysteria, and Wilbrand and Saenger are not able to report a hysterical paralysis of this nature. It is true that Gilles de la Tourette refers to *transitory lagophthalmos* during hysterical attacks, forming a part of a group, as he expresses it, of contractures and spasms difficult to analyze. Even in so-called hysterical facial paralysis, the orbicularis seems to be respected.[60]

3. **Hysterical Spasm of the Levator.**—According to Koenigshöfer,[61] hysterical cramp of the elevator of the upper lid has, up to the present time, been observed only once. No reference is given, but doubtless the statement is based upon the case which has been reported by Goldscheider, and which is summarized by Wilbrand and Saenger.[62] The patient, in addition to cramp of the levator, was affected with a cramp-like contraction of the inferior rectus, which appeared when the gaze was directed below and to the right. Parinaud has observed the simulation of Graefe's sign in exophthalmic goitre by contracture of the levator; intermittent retraction of the lids appeared even when the eyes were at rest. Carl Kunn[63] has described cramp of the levator which caused decided elevation of the upper lid and widening of the palpebral fissure.

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[60] For further discussion of this subject, see Gilles de la Tourette, Traité de l'Hystérie, I. partie, 1891, p. 415.

[61] Encyklopädie der Augenheilk., Lieferung 10, Leipzig, 1904, p. 417.

[62] Die Neurologie des Auges, Bd. i., Wiesbaden, 1900, p. 71.

[63] Deutschmann's Beiträge zur Augenheilkunde, iii. 1898.

4. Hysterical Paralysis of the Levator, or Flaccid Hysterical Ptosis.—As is well known, the existence of hysterical ptosis in the form of a flaccid relaxation of the levator was denied by Charcot and is doubted by Borel and Gilles de la Tourette. Their contention is that hysterical ptosis, as already noted, is due to spasm of the orbicularis and not to paralysis of the levator. On the other hand, equally capable observers, notably Schmidt-Rimpler, Kempner, Hitzig, Kieman, and Wilbrand,[64] have reported flaccid ptosis in hysterical subjects corresponding completely to the clinical picture of true paralysis of the levator (Fig. 11). Abadie has reported paralytic ptosis of *intermittent* form and of hysterical origin.

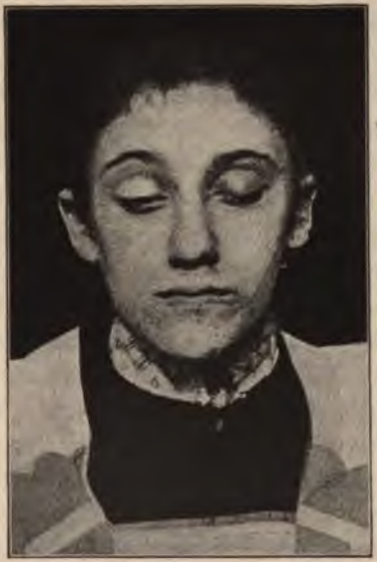

FIG. 11.—Flaccid hysterical ptosis. (After Wilbrand and Saenger.)

Undoubtedly flaccid ptosis, as compared with pseudoptosis, is rare. Usually bilateral, it has also been observed by Schmidt-Rimpler, Wilbrand, and myself as a unilateral affection. It may appear suddenly or develop gradually, and is usually seen in those individuals who exhibit other well-marked hysterical phenomena,—for example, contractures of the bulbar eye muscles, amblyopia, and amaurosis, visual field restriction, and spasm of accommodation. It may be caused by local irritation or be one of the symptoms of an attack.

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[64] For a discussion of the literature of this subject, see Wilbrand, Archiv f. Augenheilk., xxxix. 1899, p. 172, and Wilbrand and Saenger, Die Neurologie des Auges, Bd. i. pp. 463–477.

Schmidt-Rimpler regards the condition as a simple, often voluntary, relaxation of the levator, exactly as it occurs in sleep, while Hitzig ascribes it to an actual paralysis. If the development has been gradual an autosuggestive origin may be ascertained.

It is conceivable that simulation, even of unilateral flaccid ptosis, is possible, although on this point Wilbrand and Saenger have expressed serious doubts.

A differential diagnosis between flaccid hysterical ptosis and some of the forms of pseudoptosis is often difficult, and those interested in the literature and comparison of the cases should consult Wilbrand and Saenger.[65] The similarity of hysterical ptosis and the ptosis of organic disease,—for example, that of ataxia, multiple sclerosis, bulbar palsy, and even meningitis,—is often striking. To distinguish them Landolt proceeds as follows: The subject of ptosis is seated in front of the observer and commanded sharply to look at one of his eyes. To accomplish this the patient inclines the head strongly backward, so that the gaze may pass under the drooping lids. The observer now puts his hand on the top of the patient's head, who continues to regard the same fixation point, and insensibly raises and even inclines it gradually forward. If the pupils of the subject remain uncovered during this operation, hysteria may be suspected, because to effect this the lids would have to raise themselves with the eyes while the face is lowered. In other words, as Landolt expresses it, to keep the eyes open, whilst the head is inclined forward is equivalent to raising the eyes and lids when the head is straight, a thing absolutely impossible in paralysis of the levator, in which the patient ceases to see the object looked at as soon as the head leaves the compensating position, because the lids immediately cover the pupil.

Whether the so-called *morning ptosis* of Gowers, the *night ptosis* of Weir Mitchell, should be classed as a variety of flaccid ptosis is somewhat doubtful. This phenomenon will be referred to again in the section on neurasthenia, and may be dismissed now with the statement that certain nervous women, particularly on awakening in the morning, may for some minutes find it impossible to raise their eyelids without assisting the action of the levator with their fingers.

The hysterical anomalies of the external eye muscles—that is, of the bulbus musculature—next claim attention.

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[65] Loc. cit., pp. 463–477.

I.—ANOMALIES OF CONVERGENCE.

(*a*) **Convergence-Insufficiency and Convergence-Paralysis.**—The association in hysteria of insufficiency of convergence and weakness of the amplitude of accommodation has been referred to. Owing to the intimate relation existing between accommodation and convergence, if the patient makes but feeble efforts of accommodation, or, more accurately, is unable to make but feeble efforts of accommodation when looking at a near-point, a tendency to diminution of the convergence-action will appear, resulting in convergence-insufficiency, or, as it is sometimes called, a relative insufficiency of the interni. Ordinarily, under normal conditions, the amplitude of convergence is equivalent to the difference between the maximum and the minimum of convergence, or, stating the proposition in metre angles, the normal average of maximum convergence is 9.5 metre angles and the average minimum of convergence is —1 metre angle, the amplitude of convergence being, therefore, 10.5 metre angles. In hysteria this amplitude of convergence is frequently considerably reduced at both extremities. In other words, the far-point of convergence is brought nearer to the patient, while the near-point is farther removed from him, the anomaly, as Parinaud has shown, presenting the same characteristics as those which pertain to hysterical disorders of accommodation, as described on page 29. In order to determine a condition of this kind, the ordinary tests with prisms are required, diplopia not being spontaneously present. Exophoria for distance may be slight or absent, but is present at the near-point. The convergence near-point usually is less than three inches from the nose and may be removed to many inches from that point. Sometimes the patient is able to converge on an object,—for example, a pencil-point,—when brought close to the eyes, in excessive degree, but can maintain this convergence only momentarily.

The convergence-insufficiency may be so great that it constitutes a *convergence-paralysis*, a condition which has been observed in hysteria by Parinaud, Borel, and Schweigger. Although either eye can be moved inward to a normal degree when the other eye moves outward, its internal rectus does not respond to the impulse of convergence, and the convergence near-point, in place of being a few inches from the eyes, is removed to a point one or two metres from the eyes, and if the test-object is advanced within this point, insuperable crossed diplopia at once becomes manifest. A convergence-insufficiency depending upon structural or insertional anomalies of the interni, which has resulted in the development of a hysterical convergence-paralysis, may by operative interference,—for example, a tenotomy of the externus,—be followed, as Schwarz and Borel have shown, by sharp convergence-excess or spasm, which in its turn may be cured by suggestion.

(*b*) **Convergence-Excess and Divergence-Paralysis.**—An abnormal tending inward of the visual lines may depend upon excessive convergence, or deficient divergence, or upon these conditions combined.[66]

Just as a convergence-insufficiency may be replaced by an actual convergence-paralysis, so also a divergence-insufficiency may be replaced, as Parinaud, Uhthoff, Straub, von Hippel, and many other observers have noticed, by an actual *divergence-paralysis.* This condition manifests itself by homonymous diplopia and convergent strabismus when the eyes are fixed upon a distant point. As the test-object approaches the patient, and especially on lateral fixation, there is diminution of the convergent strabismus and of the diplopia, and finally a point is reached at one or two metres from the eye at which single vision and orthophoria are developed, but within this limit there is exophoria and crossed diplopia. Now this diplopia is peculiar in that, no matter whether it is homonymous on the distal side of the point at which single vision is evident, or whether it is crossed on the proximal side of this point, the images do not separate to any perceptible degree when the test-object is moved in the lateral direction from side to side. According to Parinaud, in some cases, although it is possible to obtain fusion at the point already indicated, in others such fusion is impossible, and the crossed diplopia suddenly becomes homonymous diplopia. There is, to use his phraseology, a horror of binocular single vision. This hysterical divergence-paralysis is due to spasm of convergence and not to paresis of divergence. It depends, as Kunn remarks, on cramp of the interni with atypical diplopia.

It therefore becomes evident that in hysterical patients all of the factors connected with convergence may be defective, accommodation, fusion, and probably also "the sense of convergence" (Parinaud, Hansen Grut), and we may have an absolute or a relative weakness of the adductors or a disturbance of central origin, which creates a weakness or a total disappearance of the power of fusion.

We have already seen, on page 27, that painful, hysterical visual sensations are largely connected with the anomalies of accommodation. Necessarily, however, as these anomalies are associated with those of convergence just detailed, the combination produces the highest type of so-called *hysterical asthenopia,* and while it may be true that the defects of accommodation are the predominant ones, as a rule both convergence and accommodation are involved, and there arise photophobia, retinal anæsthesia, obscuration of vision, headache, inability to maintain fixation, radiating pain, not only in the head but extending to the neck, shoulders, and chest, all aggravated by an attempt to use the eyes, but often present without ocular efforts at close ranges.

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[66] For the tests to determine these conditions, see chap. v.

Kœnigshöfer[67] thinks that in one class of cases a characteristic condition is that the patients cannot fix any one object, no matter at what distance from it they are. As soon as the attempt is made, all objects swim before their eyes, and vertigo and nausea are predominant signs. These patients direct their visual axes and accommodation upon a definite distance, so that fixation upon a nearer or further point is not possible, or possible only to a limited degree. For this form of muscular asthenopia he wishes to suggest the term "convergence immobility" (Convergenzstarre).

(*c*) **Convergence-cramp or Contracture, and Hysterical Convergent Strabismus.**—We have seen in the preceding paragraphs that in hysteria there may be convergence-excess, although it should not necessarily be regarded as an essential sign of the disease. What may be characterized as an advance on convergence-excess receives the name of *convergence-cramp*, or *spasm*, which certainly is a frequent hysterical phenomenon, and when well developed manifests itself by inward—that is, convergent—strabismus and is usually associated with spasm of accommodation.

The idea of *spastic strabismus* as part of the symptomatology of hysteria dates to the observation of Landouzy, who remarks that in the course of hysterical attacks we may notice a deviation of the eyes, to which some pathologists have applied the term *hysterical strabismus.* The earlier literature of this subject has been analyzed by Gilles de la Tourette,[68] to whose treatise those interested should turn. In more recent times detailed description of spastic hysterical strabismus will be found in the work of Borel,[69] and particularly good reports of this condition have been recorded by Manz, Schweigger, Ulrich, and Nonne and Beselin.

We have already seen that contracture of convergence, or so-called spastic strabismus, not infrequently accompanies blepharospasm, and have also noted that a condition of this character may be seen in association with hysterical ptosis,—for example, a rotation of the eyeballs downward and inward, as in Hitzig's case, followed later by convergent strabismus; inward and upward, as in a case reported by Wilbrand and Saenger; or into a position of permanent convergence, which was, indeed, never entirely overcome, as recorded by Oppenheim.

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[67] Ophthalmologische Klinik, 1902, vi. p. 113.

[68] Loc. cit.

[69] Archives d'Ophthalmologie, 1886–1887, and Annales d'Oculistique, 1897–1898.

Convergence-spasm and spastic strabismus, unaccompanied by anomalies of lid movement, in the form of blepharospasm or ptosis, occur and are characterized by convergent squint, limitation of the motility of the external recti, and by homonymous diplopia. The condition resembles paralysis of the abducens, for which it may be, indeed has been, mistaken. But, under these circumstances, double images do not behave as they would in a paralytic case. They have, as Ulrich expresses it, the same separation on looking to the right as when the patient looks to the left, a peculiarity which was exceedingly marked in a case recently under my own care, in which, with every attempt at fixation, there was excessive convergence and marked limitation of the external recti movements, but no alteration in the apparent distance between the double images, no matter to what portion of the lateral field of fixation they were moved. Schwarz insists that strict attention should be paid to the secondary deviation. If this is not greater than the primary deviation of the squinting eye, the condition is probably not one of sixth-nerve palsy, especially as one is usually able to establish in an eye of this character spastic myosis, spasm of accommodation, micropsia, and sometimes monocular diplopia. Sometimes, however, the diagnosis is extremely difficult, as Schwarz himself admits, as is evident from an examination of the case reported by Nonne and Beselin. Schwarz believes that the only positive differential diagnostic point between a convergence-cramp and an abducens paralysis is developed by an objective investigation of the optical apparatus. In a case of his own he was able to determine by a skiascopic examination that in one direction of the gaze at which the double images were evident an accommodation of more than 2 D. developed. Nonne and Beselin point out that in spasm, in contradistinction to palsy, contractions of the muscles innervated by the facial, dread of light and pain in and around the affected eye would be apt to be present.

Hysterical spastic strabismus, or, more accurately, as Parinaud properly insists, contracture of convergence, may occur as the prodrome of an attack of grave hysteria, during hysterical attacks and at their termination, but is, in general terms, a transitory phenomenon, although it may terminate in some form of deviation,—for example, a conjugate deviation,—exactly as a conjugate deviation may sometimes precede a convergent strabismus. Such contracture of convergence, or strabismus, may arise in association with other muscular movements—as, for example, in Shuster's patient with traumatic hysteria, in whom when the arm was raised strabismus developed. It can be brought about, as other hysterical phenomena, by suggestion and cured in like manner. Permanent convergent strabismus in hysteria must not be regarded as a stigma of the disease. An investigation will always show other factors upon which this depends, notably hyperopia, anisometropia, amblyopia of one eye, and absence of the fusion sense. In a notable case under my own observation of long-standing major hysteria, to which reference will again be made, a prolonged conjugate deviation of the eyes to the left lasting for many months was followed upon recovering by well-marked convergent strabismus, occurring, as is usually the case in hysteria, in crises, but finally settling to a well-marked moderate inward strabismus. When investigation was thoroughly made a high degree of hyperopia, with all of the symptoms of convergence-excess, was found, and suitable glasses produced parallelism of the visual axes.

While it is true that in the convergent strabismus of the character which has been described as hysterical there may be limitation of the outward movements of the eyes, it should be remembered that this is not always the case, and that one sign in the minor degrees of convergence-contracture which helps to differentiate it from an abducens palsy is that the field of fixation is normal in the one and naturally limited in the other. When the convergence contracture is excessive, then this test cannot be applied, although it does not in the least destroy the very evident etiology of the affection as an anomaly of convergence innervation.

Spasmodic Conjugate Deviation.—In discussing spastic strabismus in hysteria, we have seen that there may be a spasm or cramp of an individual muscle, or in association with blepharospasm, or various types of ptosis, and have noted in detail the well-marked hysterical phenomena of convergence-cramp and a strong inward strabismus, often combined with spasm of accommodation. We have now to deal with varieties of conjugate deviation, in which a spasmodic condition of associated muscle groups is evident. During clonic or tonic hysterical convulsions there may be marked contraction of the ocular muscles and with this a coincident contraction of the muscles of the neck, giving the appearance of a *conjugate deviation of the head and eyes*, or, again, under similar conditions, the eyes may be turned in any direction by what Pansier calls convulsive rollings, without any apparent rule guiding the irregularity of the spastic rotations. These are not true forms of spastic strabismus. Both eyes execute incessant movements but are not permanently deviated. They seem, to use the language of Parinaud, to have lost the power of fixation. Often these cases may be complicated with photophobia and blepharospasm, and there may be lagophthalmos owing to a contracture of the levator palpebræ, as pointed out by Levy and described by Gilles de la Tourette and Parinaud. Such irregular contractures of the ocular muscles may be accompanied by diplopia, or, more accurately, by attacks of diplopia, occurring sometimes frequently during a single day.

In typical spasmodic conjugate deviation the contractures affect the *associated parallel movements*, or, as Parinaud puts it, the *associated movements of direction*, and while it is true that the deviations may be in any direction, according to the same author, the inferior and oblique position is preferred. As may have been inferred, the condition is not commonly a permanent one, occurring as it does during seizures, and yet what may be designated permanent deviations of well-marked character have been recorded. Notable in this respect is the case of Frost,[70] in which the eyes were directed downward and to the right, upward movements being almost impossible, and the condition lasted one year. I studied with Dr. Wharton Sinkler, of this city, a case of major hysteria in which there was pronounced unvarying conjugate deviation upward and to the left, with lagophthalmos, owing to contracture of the levator palpebræ of each eye, which lasted for nine months. In less persistent cases there may be attacks, lasting for days or weeks, coming on with other convulsive seizures; or the type may change—that is, a conjugate deviation in one attack may become a spasm of convergence in another, as I myself have noted in the case already referred to, and as has also been reported by Mann. The combination of contractures of convergence with loss of lateral movement has already been described, and typical cases of this character are not uncommonly seen.

Ordinarily the contractures upon which depend these conjugate deviations necessarily indicate the implication of both eyes simultaneously, but it should be remembered that in unilateral blepharospasm there may be, when the patient attempts to fix the affected eye, a prompt spasmodic deviation upward and inward. This unilateral condition of spasm is illustrated in Goldscheider's case, whose patient exhibited nothing peculiar in the rotations of the ocular muscles until she was required to look to the right and below, when the left bulbus was spasmodically drawn downward and inward, while the left upper lid was elevated. These spasms of the ocular muscles, to which Borel makes full reference, have already been discussed under spastic strabismus. As in other hysterical ocular phenomena, with these conditions may be present amblyopia, photophobia, and blepharospasm, and they may be brought into existence by emotion, trauma, intoxication, or reflexly by dental irritation, and would seem to be more often found in young hysterics, who, curiously enough, apart from their convulsive seizures, may have little marked other stigmata of hysteria.

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[70] British Medical Journal, 1884.

Hysterical Palsies of the Ocular Muscles: Hysterical Ophthalmoplegia.—(*a*) **Palsies of Individual Muscles.**—Binswanger,[71] referring to this much disputed subject, gives a summary of what he calls the approximately authentic cases of hysterical paralysis of the eye muscles, as follows: Paralysis of the left internal rectus and the superior recti muscles on both sides (Guttmann[72]); paralysis of the right external rectus, later of the left (Duchenne[73]); bilateral paralysis of the abducens (Leber[74]); paralysis of the abducens (Parinaud[75]); paralysis of the right and later of the left abducens combined with contracture of the internal rectus (Borel[76]); ptosis, paralysis of accommodation, paralysis of the internal rectus and of the inferior rectus of the right eye (Parinaud and Richer[77]); paralysis of the abducens, two cases (Wilbrand[78]); paralysis of the left inferior oblique (Wilbrand and Saenger, examined later by Nonne and Beselin[79]). In the last named case paralysis of the oblique on the left side was made to disappear by suggestion, and so-called hysterical paralysis of the right superior oblique was later observed. Other cases, not quoted in this summary, are these: Ptosis, divergent strabismus, and nystagmus, especially occurring in children (Steiner[80]); palsy of all eye muscles except the internal recti (Stephenson[81]); double oculomotor palsy of hystero-traumatic origin (Debove[82]); ptosis with complete fixation of the left eyeball (Cowan[83]). Schäfer's assertion that oculomotor palsy is frequent in children has not been confirmed, and Binswanger regards it as erroneous. It is hardly necessary in an article of this character—indeed, space does not permit—to analyze the case histories. Such analyses have been well made by Binswanger, Möbius, Parinaud, Kunn, and many others, and it would seem to be settled with a fair amount of definite accuracy that, as Koenig has said, thus far it does not seem that any true case of hysterical eye-muscle palsy has been published.

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[71] Loc. cit., p. 389.

[72] Berlin. klin. Wochenschr., 1869.

[73] Gaz. des Hôp., 1875.

[74] Graefe u. Saemische, Handbuch der gesammten Augenheilk., I. Aufl., v. p. 985.

[75] Soc. d'Ophthal. française, 1886.

[76] Arch. d'Opth., 1886.

[77] Binswanger, loc. cit.

[78] Jahrb. der Hamburger Staats-Kranken-Anstalten, 1890.

[79] Binswanger, loc. cit.

[80] Jahresbericht f. Ophthal., xxix. 1898, p. 567.

[81] British Medical Journal, 1902, i. p. 205.

[82] Recueil d'Ophthalmologie, xiii. 1891, p. 124.

[83] New York Eye and Ear Infirmary Reports, January, 1901.

Furthermore, as Gilles de la Tourette has expressed it, we may suggest to a patient palsy, but cramp or contracture always appears. Parinaud, to whom we owe so much for his accurate study of hysterical cases, and who has himself reported palsy of ocular muscles in hysterical patients, remarks: Paralysis of the third pair and contracture limited to one muscle, or to the territory supplied by one nerve, probably do not exist in hysteria, but he further goes on to say, as hysteria is a disease continually presenting new phases, it is prudent to make a reservation that such a condition may have been observed and implies that it may in the future be observed. He quotes Samelsohn as having seen one case of hysterical paralysis of the sixth pair, but disputes Borel's case of abducens paralysis, which he thinks on careful analysis indicates a condition to be explained by the symptom already so much dwelt upon, namely, contracture of convergence. He feels sure that any case of well-marked paralysis of the third pair in a hysterical patient—for example, one that he himself has reported which he observed for a number of years—should give rise to the suspicion of another influence and of the coexistence of two separate diseases.

How, then, are these conditions to be explained? Binswanger holds that the so-called hysterical paralyses of the eye muscles always indicate a defect of the associated lateral movements, or a motor insufficiency of certain muscular movements closely connected with psychical processes, and sometimes a combination of paretic and spastic motor disturbances peculiar to hysteria. I have never seen, in a very large experience in hysterical patients, any isolated paralysis of an eye muscle, and believe with Parinaud and other writers that the so-called hysterical palsies should be explained by what is sometimes described as an associated ocular paralysis,—that is, although the eyes cannot make certain movements in which they are usually associated, the directing power of the muscles may be unimpaired when they exercise their function in a different direction. In other words, there is a paralysis of movement and not of the muscles supplied by a given nerve. We have, then,—

(*b*) **Palsy of associated parallel movements,** or, in other words, the Blicklähmung of the Germans. Thus, as, for example, in a case of Claude's, there may be inability of the eyes to move to the left,—that is, loss of the directing power of the right internus and of the left externus, which are associated antagonists; there is loss of lævoversion, using Duane's phraseology. In the same way there may be loss of dextroversion, and so on, of the several different associated parallel movements.

(*c*) **Hysterical ophthalmoplegia**, many cases of which are on record, as Parinaud has well pointed out, is characterized essentially by the fact that the voluntary eye-muscle movements are affected, and the patient cannot turn or rotate the eye to any designated point, although the same movement can usually with ease be involuntarily accomplished. The condition has been compared to the characteristics of mobility of the lower extremity in hysterical abasia. For example, at present I am studying with Dr. Wharton Sinkler, of this city, a case of pronounced hysteria in which the patient cannot raise the eyes in the least above the horizontal level when directed to do so, although she is perfectly able when her attention is directed momentarily away from the thought of upward rotation to make this movement. A similar associated paralysis of the superior recti muscles is well described by Nogues and Sirol.[84] If the upper lids are raised, the upward rotation of the globe above the horizontal plane is impossible, although all other associated movements are preserved. The movement of elevation, which cannot be excluded when the lids are elevated, can be made when the lids are closed. It is not necessary to multiply instances. Even in cases of conjugate deviation it is possible—as, for example, it was in Frost's case, in which both eyes were directed downward and to the right—to make the one eye fix perfectly and follow the movements in all directions when the other eye was closed, and the same observation has been made by Parinaud in one of his patients. Furthermore, these so-called palsies disappear during chloroformization.

Cases of *hysterical ophthalmoplegia* have been described—for example, by Ballet, Warner, Bristowe, and others—in hysterical patients the subjects of exophthalmic goitre, but even here—as, for example, in Ballet's case, although there was complete inability to move the eyes, which were normally directed—automatic movements were possible. Raymond and Koenig, referring to complete hysterical ophthalmoplegia characterized by complete loss of voluntary movement, decline to regard them as true paralyses and insist on a differential diagnostic point between this condition and a genuine ophthalmoplegia in the absence of mydriasis. Nonne and Beselin quote cases of palsy of the external eye muscles lasting for several days, accommodation and pupillary functions being normal. These may be explained by assuming a transitory spasm of the external eye muscles, which, according to Gilles de la Tourette, are common in hysteria and may produce, as has already been pointed out, temporary attacks of diplopia.

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[84] Annales d'Oculistique, cxxxv. 1901, p. 360.

(*d*) **Dissociation of the Eye Movements.**—According to Kunn,[85] many cases of so-called palsy and cramp of the eye muscles in hysteria are really dissociations of the eye movements. This condition is characterized, according to him, not by a lasting and constant limitation of movement, but by a loss of movements or rotations that were formerly associated, and takes place in both eyes, so that exactly in the atypical and changing condition of the rotation disturbances the characteristic symptom of the condition is contained. Borel has described in hysterical patients, and by ingenious experimentation proved, a loss of the muscular sense of the eyes.

Hysterical Nystagmus.—Binswanger maintains that a genuine hysterical nystagmus lasting for some time and occurring independently of hysterical paroxysms does not occur. Pansier remarks that just as nystagmus is frequent in epilepsy it is rare in hysteria. He quotes Rouffinet as attributing to it the following characters: It is mixed,—that is to say, it exists as well in a state of fixation as in a state of non-fixation; it is unaccompanied by refractive errors; it is permanent and its intensity can diminish with the disappearance or the diminution of the crisis; but it does not disappear completely and differs from that seen in multiple sclerosis, inasmuch as the latter is exaggerated by the movements of the ocular globes. As is well known, Charcot insisted that nystagmus served as a differential diagnostic symptom between disseminated sclerosis and hysterical affections, being present in the former and absent in the latter. Sabrazes thinks he has proved that nystagmus should be regarded as a possible hysterical manifestation because of the efficacy of suggestion in one of his cases. This inhibited the nystagmic movements, and there were in addition the presence of other hysterical phenomena. Oscillation or nystagmic movements of the eye may be produced by hypnotic suggestion in hysterical patients. A rapid oscillation of the eyeball, either in a horizontal or oblique direction, synchronous with contractions of the orbicularis when resistance is offered to its contraction with the fingers, has been recorded by Stransky and is described by him as an associated nystagmus. It occurs, according to this author, in neurotic individuals. Santos Fernandez[86] has reported hysterical nystagmus. The rapid and sometimes bizarre movements of the eyeballs which occur during hysterical attacks, and to which reference has already been made, are well known and cannot be regarded as examples of true nystagmus. (Compare Chapters V and IX.)

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[85] Deutschmann's Beiträge f. Augenheilk., vol. iii. 1898.

VII. PREVIOUSLY UNCLASSIFIED HYSTERICAL OCULAR PHENOMENA.

1. Disturbances of Sensation.—Briquet considered anæsthesia of the conjunctiva, especially of the left side, as a condition so constantly present in hysteria that it might be regarded as characteristic. Schwarz, however, maintains that conjunctival and corneal anæsthesia is not peculiar to hysteria, but is seen in pure neurasthenic cases. This is contrary to my experience. The entire conjunctival expansion may be insensitive, or the anæsthesia may be confined to certain areas, particularly to an area below the cornea. The tarsal conjunctiva is less frequently anæsthetic than the bulbar mucous membrane. The cornea, more uncommonly than the conjunctiva, is involved in anæsthesia. According to Gilles de la Tourette, this is usually incomplete, the centre of the cornea retaining its sensibility. Féré, as already noted, believed that hysterics with hemianæsthesia unassociated with restriction of the visual field and achromatopsia did not lose the sensibility of their conjunctivas, but if they had dyschromatopsia and concentric contraction of the field of vision the conjunctiva would be anæsthetic, while if there was achromatopsia and almost complete loss of indirect vision, both the conjunctiva and cornea would be insensitive. This observation has not been confirmed by other observers. The semeiological value of corneal and conjunctival anæsthesia in hysteria has recently been discussed by Robinau.[87]

In moderate grades of conjunctival anæsthesia the *palpebral reflex* is retained, while in higher grades it is lowered or lost. It may be lost, too, if the cornea, being anæsthetic, is touched, but if the test-object comes into the field of the pupil there is a response. This, however, is not due to contact, but is a *retinal lid-closure reflex*. The *lachrymal reflex* is retained even when the conjunctiva is wholly insensitive, as has been shown by Pitres, Gilles de la Tourette, Spiller, and others. This preservation of the glandular reflex, as Spiller suggests, may serve to distinguish hysterical from organic anæsthesia. In place of anæsthesia of the conjunctiva and cornea there may be *hyperæsthesia* of these membranes and often associated with it amblyopia, lachrymation, photophobia, and orbicularis spasm.

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[86] Clin. Opht., Paris, 1904, x. 345.

[87] Bordeaux Thèse, 1904.

Anæsthesia of the skin of the lids and surrounding cutaneous surface is also present in hysterics, but not so frequently as the same condition of the oculo-mucous membrane. As before noted, Gilles de la Tourette has described a periorbital zone of anæsthesia in non-painful hysterical blepharospasm, which in the painful variety of the affection is replaced by a similar hyperæsthetic area.

Spots sensitive to pressure in the neighborhood of the eyes are common in hysteria, especially along the inferior orbital border. These tender points Schwarz considers to be quite characteristic of hysteria in the absence of neuralgia, and if they do not represent the position of a nerve-trunk or one of its points of exit.

Hysterogenetic zones in and around the eye—for example, in the infra- and supraorbital region—have been described, and Lichtwitz has recorded a case where the conjunctiva, cornea, and lachrymal canals were occupied by hypnogenic, letharogenic, and spasmogenic zones, interference with which produced conditions indicated by their descriptive names.

2. **Disturbances of Secretion.**—Excessive secretion of tears, sometimes paroxysmal in nature, is frequent in hysterical patients, and is sometimes called *hysterical epiphora*. It is often accompanied by spasm of the orbicularis. Bloody tears—*i.e.*, bleeding from the outer angles of the eyes—and the so-called vicarious menstruation have been described by Hensinger and others in subjects of hysteria (page 342).

Chromidrosis, which consists of a bluish-black discoloration of the lids, somewhat oleaginous in nature, and which can be wiped away, is occasionally encountered in hysterical subjects, but is not necessarily a stigma of the disease. Hysterical chromidrosis lasting for months has been described by Putnam. Severe cases of oily seborrhœa have been reported in which the dusky secretion has been allowed to accumulate for several years, and in a remarkable example of this nature recorded by Van Harlingen[88] a thick black mask of the face, covering the forehead and nose, had been allowed to form.

3. **Chromatopsia.**—Strzeminski[89] has reported *erythropsia* as a rare phenomenon in hysteria and refers to similar observations by Hilbert. Fano had a patient who saw in the field of vision spots of color, and Meirowitz[90] one who when looking into the air detected colored

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[88] American Journal of the Medical Sciences, '04, p. 245.

[89] Recueil d'Ophthalmologie, 3 Series, 1902,

[90] Journal of Nervous and Mental Diseases.

concentric rings, processions of blue commas surrounded by green spots, while objects and persons were often surrounded by colored halos. I have recorded[91] a case of hysterical amblyopia associated with toxic amblyopia in which a marked symptom was xanthopsia. Ordinary non-chromatic visual hallucinations are met with in hysterics. Binet and others have shown that tactile sensations may be transformed in visual sensations in hysterical subjects.

4. **Organic Changes.**—An essential condition of hysterical amblyopia and amaurosis is the absence of organic lesions in the fundus of the eyes. Hyperæsthesia of the retina may be present; enlargement of the retinal veins and serous exudate into the retina have been recorded by Landolt. Leber and Nuel have described hyperæmia of the retina and papilla, and a gray discoloration of the nerve-head which may eventuate in atrophy. But, as Nuel himself remarks, in such cases some other agency should be suspected; the hysteria may well have been associated with multiple sclerosis or tabes dorsalis. The hysterical irido-choroiditis described by Simi is open to doubt. Hysterical amblyopia may simulate sympathetic disease, as has been well shown by Parinaud and Kalt.[92] The latter author observed a young woman whose left eye had been destroyed by irido-choroiditis. There was depreciation of vision of the right eye, restricted fields for blue, and red and green achromatopsia. All of these symptoms disappeared when the left eye was enucleated. Rapid opacity of the crystalline lens after an hysterical attack has been reported by Masleunikow.[93] He attributes the condition to nutritional disturbances caused by repeated cramp of the ocular muscles. An hysterical vasomotor affection of the conjunctiva has been described by Fromaget[94] in the form of intermittent congestion of the bulbar conjunctiva without inflammatory phenomena. Hysterical œdema of the lids is probably always the result of some inflammatory condition of the conjunctiva or ciliary margin, and not an essential symptom of the disease. Some hysterics purposely bring irritating substances in contact with their eyes to cause congestion and excite sympathy, and may even, as did one of my patients, deliberately scratch their corneas with pins. Hysterical girls sometimes pull out their eyelashes, and to the result of this performance Gifford has given the name "hysterical alopecia of the eyelids."

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[91] Ophthalmic Record, viii. 1899, p. 18.

[92] Annales d'Oculistique, vii. 1892, p. 37.

[93] Zeitschr. f. Augenheilk., v. 1901, p. 477.

[94] Recueil d'Ophtal., 3 Series, 24, 1902, p. 248.

CONCERNING THE ETIOLOGICAL FACTORS IN HYSTERICAL OCULAR STIGMATA.

In general terms the psychoneurosis to which the name "hysteria" usually is applied is responsible for the development of the ocular signs which have been described, no matter which of the causes of the great neurosis may have been originally operative. As next to heredity trauma takes position as a cause of hysteria, it is not unnatural that many eye-symptoms of this disease form part of the symptomatology of *traumatic hysteria*. It is not germane to the subject to discuss the distinction, if it exists, between *hysteria* and *traumatic hysteria*, to some phases of which, as is well known, under the name *traumatic neurosis*, Oppenheim and Thomsen have given a special position in the nosological index, except to say that the ocular anomalies of both are analogous, if not identical, and in this respect, at least, if not in all others, the two affections are also identical. In the eyes the manifestations of hysteria which have been described as hysterotraumatic are often pronounced,—more so, frequently, than those which are associated with hysteria of other origins,—but they are not pathognomonic. In other words, given certain ocular stigmata of hysteria, one could not expect them to reveal the original cause of the hysteria,—that is to say, whether it depended upon heredity, trauma, disease, defective mentality, toxæmia, etc.,—although they might vary in their severity and endurance. The eyes and, in general terms, the optical apparatus contain the necessary structures through which the well-known signs of hysteria—disturbances of sensation, spasms and contractures—may become evident in a manner similar to their development in other organs and regions of the body.

To the disturbances of the functions of the eyes we are often able to apply tests more delicate than those which pertain to the investigation of other organs, and therefore should be able more accurately to interpret the symptoms. The admirable investigations of Landolt, Galezowski, Parinaud, Gilles de la Tourette, Pansier, Borel, Wilbrand, Schmidt-Rimpler, and Schwarz are of especial value in the thoroughness with which their studies have been pursued from these and all other stand-points.

Unfortunately, however, in many of the detailed journal reports of hysterical ocular phenomena the lack of ophthalmological training is only too evident, and especially regrettable is the absence of proper investigation of the refraction of the eyes. Thus, Dejerine points out that in many cases of so-called unilateral hysterical amblyopia it is quite possible that the one eye may have been emmetropic, or approximating that condition, while the other may have lost visual acuity owing to refractive anomaly which has wrongly been attributed to hysteria. Pansier and Lapersonne insist on the difficulty of distinguishing between hysterical spastic convergent strabismus and convergent strabismus associated with hyperopia in an hysterical subject. The effect of hyperopic refraction on the anomalies of accommodation so commonly observed in hysterics must be evident, and Parinaud has not failed to call attention to it in his beautiful studies of this subject, and to the modifications which must necessarily be produced by myopia and presbyopia. What the relations of the various phases of exophoria and esophoria must be to the hysterical anomalies of convergence is only too plain to the student of ocular muscle imbalance. In a word, there is constant danger of attributing to hysteria an anomaly of ocular function, when really this is a condition belonging to the eyes of an hysterical subject. More than this,—and it has been strangely neglected in the investigation of hysterical phenomena,—refractive error, especially astigmatism, muscular imbalance, in short, "eye-strain," are responsible for many cases of hysteria which manifest themselves by well-known stigmata, not only in the eyes but in other regions and organs of the body, or by the phenomena pertaining to seizures. It may perhaps be doubted whether refractive error and heterophoria produce hysterical manifestation in individuals of sound constitution and normal resisting power, but they certainly can call them into existence in those of neuropathic tendency. Given a normal subject with moderate hyperopic astigmatism and esophoria (convergence-excess), and the eye troubles will interpret themselves by headache, relieved by their correction, or perhaps resisted without serious detriment for long periods without correction. In a neuropathic subject with the same defects their interpretation is likely to be spasm of accommodation, anæsthesia of the retina, contracture of convergence, restriction of the visual field, and areas of anæsthesia often relieved by their correction, but in the absence of it their influence is unresisted, and the long-enduring phenomena of what may be called a general hysteria result.

For the treatment, see page 74.

OCULAR MANIFESTATIONS OF NEURASTHENIA AND TRAUMATIC NEUROSES.

Although the disturbance of the visual sensation to which neurasthenic patients and those who are the subjects of traumatic neuroses are liable has been much investigated, it cannot be stated, as it was of [illegible], that [illegible] ocular signs as have been described constitute a distinct symptom-group. For convenience, the following subdivisions of [illegible] may be made:

(1) Inferior and painful visual sensation, or neurasthenic—more properly nervous—asthenopia.

(2) Anomalies of the iris movements, or neurasthenic pupil phenomena.

(3) Anomalies of the eyelid and eye-muscle movements, or so-called neurasthenic insufficiencies of the ocular muscles.

(4) Anomalies of the visual field.

(5) Previously unclassified neurasthenic ocular phenomena.

Neurasthenic or Nervous Asthenopia.—Certain systematic writers, for example, Wilbrand and Saenger,[illegible]—have made a distinction between nervous asthenopia, or the insufficiency of visual power seen in nervous persons, as it occurs in school children and during the years of early development, and as it appears in adults. School children, particularly from the ninth to the fifteenth year, often are brought for examination because they complain of a flickering before the eyes,—letters and figures disappear as they look at them and the print runs together, and reading creates lachrymation and severe brow and eye pain. When the child is asked to read he gradually approaches his face to the book, moves his head from side to side instead of following the lines with his eyes, and tries to bring to his aid stronger and stronger light. In association with the difficulty of vision are contraction of the eyebrows, half-closing of the eyelids, and often twitching of the face and muscles of the shoulder, or, in other words, the symptoms of the so-called habit spasm. This gradual approaching of the book towards the face, so that the patient is able to obtain larger retinal images, is more or less characteristic of this condition in childhood and is not seen in the adult cases. It depends upon a cramp of accommodation, and when the cramp subsides and there is tiring of the accommodative muscle there is corresponding blurring of the print and running together of the lines. If the visual power is tested in the ordinary manner with test-types, it will usually be found to possess a depreciation greater than would be accounted for by any error of refraction, and will often be promptly raised to normal if the letters are viewed through tinted glasses. In other cases glasses fail to improve the visual acuity.

Associated with this asthenopia and diminution of visual acuity

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[illegible] Ueber Sehstörungen bei functionellen Nervenleiden, Leipsic, 1892, p. 58.

concentric contraction of the visual field may be noted, in the manner which will be described in a subsequent paragraph.

It is noteworthy that in children these ocular phenomena are marked to the exclusion of other neurasthenic phenomena, while in adults the reverse may be the case. The subjective symptoms in adults are similar to those just detailed. They have been described by Wilbrand, Standish, and others somewhat as follows: Headache, particularly throbbing in the brow and temples, occipital distress, pain in the back of the neck and spine, vertigo, muscæ volitantes, defective accommodation, intolerance of light, and improvement in vision in the dusk and through tinted glasses. In addition to this, any attempt at concentrated vision is followed by a rapid disappearance from view of the object which is to be fixed. There are diminution of central vision, sudden attacks of obscuration of vision, processions of scotomas, visual hallucinations, lack of fixation of the optical memory images, persistent and confusing after-images, colored vision,—for example, erythropsia,—and a sudden red color of the pages of a book while the letters seem to be green. Associated with these phenomena are the characteristic changes in the visual field presently to be described.

Etiology.—While it is true that neurasthenic patients, both children and adults, may possess as part of their symptomatology the symptoms of defective and painful visual sensation which have been detailed, nervous asthenopia, as Schwarz[96] properly remarks, is justified as a descriptive term only when the chief symptoms of asthenopia—namely, hyperopia, astigmatism, and ocular muscle imbalance—have been excluded. In other words, as is now well known but as evidently was not well recognized by earlier writers, these patients have nervous asthenopia not because they are neurasthenics, but because they have various refractive errors, particularly astigmatism, and various types of muscular imbalance, which, indeed, are in many cases the causative factors of the neurasthenia itself, and all of the symptoms, those referable to the eye as well as to the general economy, are simply the manifestations of eye-strain occurring in individuals of neuropathic constitution. They appear in these bizarre manifestations, because of such constitution, while in another individual of sound health they might either be resisted or find interpretation in a milder type of so-called asthenopia.

Anomalies of the Iris Movements, or Neurasthenic Pupil Phenomena.—Unlike hysteria, there are no pupil phenomena which in any sense are characteristic of the neurasthenic state. It is true, as Beard pointed out, that the pupils are sometimes unequal, but not more frequently, as Schmidt-Rimpler[97] maintains, than has been observed under physiological conditions. Wilbrand also declares that the pupil reactions are usually normal, and that the slight differences in the width of the pupil which are often seen in these individuals depends upon certain conditions of the eye itself, and not upon the general and physical state. The inequality of the pupil observed in neurasthenia, according to Dercum, is always transient in character. It is true that the pupils of neurasthenics are often wider than those of more stable physical condition, and they sometimes seem to manifest an unusually prompt response to light impulse. On the other hand, occasionally they also act less promptly to light.

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[96] Die Bedeutung der Augenstörungen für die Diagnose der Hirn- und Rückenmarks-Krankheiten, Berlin, 1898, p. 97.

Hippus is said to be more common in neurasthenia in an exaggerated condition than in other functional nervous states, but this is doubted by many competent observers, and my own observations of many neurasthenics would not lead me to believe that hippus should be regarded as a sign of neurasthenia, nor have I ever seen any pupil change which is significant of a pure neurasthenia. Should there be reflex immobility to light in a patient exhibiting the symptoms of neurasthenia, there would be good reason to suspect tabes or beginning paresis.

Anomalies of the Eyelid and Eye-Muscle Movements, or So-called Neurasthenic Insufficiencies of the Ocular Muscles.—In neurasthenia, unlike hysteria, anomalies of the musculature of the bulbus and of the lids are for the most part wanting. Certainly the various types of contracture of the eye muscles are lacking, nor is blepharospasm, as it has been described in connection with hysteria, present. A frequent symptom in neurasthenia is an inability of the patient to close the eyes completely when standing with the legs together (Romberg's sign), associated with marked fibrillary twitchings of the orbicularis. This lid phenomenon has sometimes been called *Rosenbach's*[98] *sign*, which he and Banas have considered as a characteristic symptom of neurasthenia, but which Schmidt-Rimpler declines to regard as peculiar to this affection, inasmuch as it is often seen in healthy individuals. According to some writers,—for example, Loewenfeld,—this symptom is also present when the legs are separated or when the patient is in the sitting position. It has been ascribed to weakness of the orbicularis and to spasm of the sympathetic. As Knies puts it, the voluntary motor innervation is enfeebled and the sympathetic reflexes are increased.

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[97] Die Erkrankungen des Auges im Zusammenhang mit anderen Krankheiten, Wien, 1898, p. 276.

[98] Centralblatt f. Nervenheilkunde, ix., No. 17.

According to Landolt,[99] there is a form of insufficiency of convergence which accompanies the neuroses, and especially neurasthenia, to which he has applied the term *neuropathic insufficiency of convergence,* and which he describes as follows: The excursion of the eyes may be normal, the action of the abductors—that is, of divergence—also normal or even augmented, but adduction is deficient. In such cases there is a reduction at both ends of the range of convergence. (See also article on hysteria, p. 44.) Landolt believes that this insufficiency of the innervation of the adductors, or of convergence, is a phenomenon which should be regarded as analogous to the weakness of the orbicularis which has just been described as Rosenbach's symptom.

A symptom often noted in neurasthenic as well as hysteric patients (see section on hysteria, p. 656) is the so-called *morning ptosis* of Gowers, the *night ptosis* of Weir Mitchell, and which has been regarded by some authorities as a form of flaccid ptosis, which is, as has previously been noted, a frequent sign in hysteria. Certain nervous individuals, particularly women, usually on awakening in the morning, or during the night, find it impossible to raise their eyelids without assisting the action of the levator with their fingers. There is, in other words, a sensation as if the lids were glued together, and associated with it there is often a dry catarrh of the conjunctiva or a hyperæmic condition of this membrane. I have studied a number of cases of night ptosis of this variety, and all of its subjects were nervous persons in the ordinary sense of the term and not hysterics. Usually they have been women past middle life, who have often been seriously alarmed, thinking that they had suddenly become the subjects of a paralysis. In most instances proper general regimen, with correction of existing refractive errors, has relieved the difficulty. Nystagmus has been described. According to P. C. Knapp, strabismus and nystagmus may be present in certain traumatic neuroses, but they are really traumatic scleroses.

Anomalies of the Visual Field.—Of greater interest than the other ocular symptoms associated with neurasthenia are those changes in the field of vision which interpret the ready exhaustion to which the visual apparatus of the subjects of this condition are liable. They have been elaborately and especially studied by Förster,[100] Wilbrand and Saenger,[101] Wilhelm Koenig,[102] Peters,[103] Frank-Hochwart and Topolanski,[104] Schmidt-Rimpler,[105] von Reuss,[106] H. Arnheim [107] and L. Wolffberg.[108] To the writings of these authors I am much indebted in the preparation of the following pages.

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[99] The Refraction and Accommodation of the Eye and their anomalies. Philadelphia, 1886, p. 504. A System of Diseases of the Eye, edited by Norris and Oliver, vol. iv. p. 136.

In general terms these alterations in the visual field may be described as fatigue constrictions or contractions, and, according to Wilbrand and Saenger, the contraction may extend to the point of disappearance of the visual field, or, more usually, to a certain point of concentric narrowing. Furthermore, the fatigue phenomena may appear in all of the four halves of the visual field or only in certain ones. According to the same authors, the signs of fatigue may manifest themselves when the extent of the visual field and central visual acuity is at first normal, or when the extent of the visual field is at first normal, but visual acuity is diminished, or, finally, when the extent of the visual field is concentrically contracted from the beginning and central visual acuity is diminished.

Von Reuss insists that as neurasthenia is characterized by an easy exhaustion of the visual apparatus, this gives rise to visual fields of a fleeting and changing nature. The diminution of the size of the field appears, according to him, only during the process of examination, or, if it is present from the beginning, it is due to exhaustion of the eye by a previous examination. Systemic writers have described certain types of visual field fatigue as follows:

I. **Concentric Contraction.**—There may be a general concentric contraction of the visual field similar to that seen in hysteria, but differing from it, according to von Reuss, because in neurasthenia it is constantly changing during examination, while in hysteria it does not alter its size until recovery ensues. Moreover, there is no change in the sequence of the color circles.

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[100] Bericht über die zehnte Versammlung der Ophthalmologischen Gesellschaft, Heidelberg, 1877, p. 162.

[101] Ueber Sehstörungen bei functionellen Nervenleiden, Leipzig, 1892, and Wilbrand, Die Erholungsausdehnung des Gesichtsfeldes unter normalen und pathologischen Bedingungen, Wiesbaden, 1896.

[102] Ueber Gesichtsfeld-Ermüdung und deren Beziehung zur concentrischen Gesichtsfeldeinschränkung bei Erkrankungen des Centralnervensystems, Leipzig, 1893. (This work contains a full bibliography.)

[103] Deutsche Zeitschrift f. Nervenheilk., Bd. v., 1894, p. 302.

[104] Deutschmann's Beiträge zur Augenheilk., ii., 1895, p. 46.

[105] Die Erkrankungen des Auges im Zusammenhang mit anderen Krankheiten, Wien, 1898, p. 266. (This work contains an extensive bibliography.)

[106] Das Gesichtsfeld bei functionellen Nervenleiden, Leipzig and Wien, 1902.

[107] Fortschritte der Medicin, vol. xx., 1903, p. 44. (This paper contains a critical review of the subject with an analysis of the important papers.)

[108] Archives of Ophthalmology, xxxiii., 1904, p. 597.

Temporary uniform concentric contraction of the visual field, so that it resembles a circle, or closely approaches in appearance a reduced visual field, frequently follows an hysterical, hystero-epileptic, or epileptic attack, and hence is not peculiar to neurasthenia.

Permanent uniform concentric contraction of the visual field is not a neurasthenic phenomenon, unless those examples which Wilbrand has described in certain types of traumatic neuroses be excepted.

II. **Förster's Shifting or Displacement Type of the Visual Field.**—Perimetric examination of certain nervous patients will often yield a visual field of very irregular type, due to a more or less unsymmetrical contraction of the field from meridian to meridian brought about by fatigue. This is the important symptom of that condition called by Von Graefe "anæsthesia of the retina," but now generally known by the name of "nervous asthenopia," proposed by Wilbrand in place of the more restricted term "neurasthenic asthenopia," and the general visual symptoms of which have already been described. (See Page 672.)

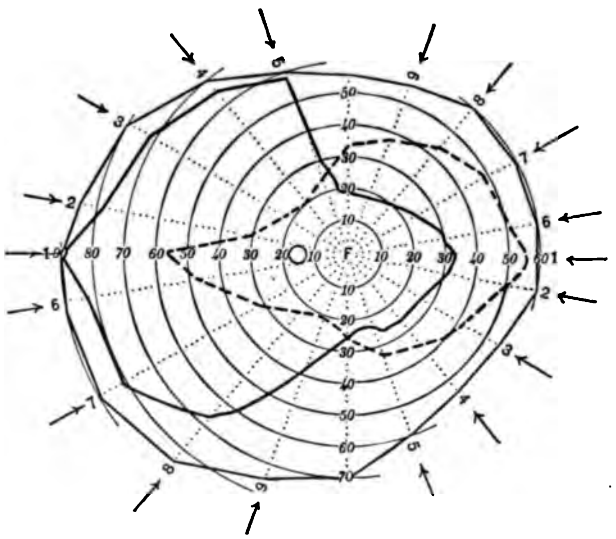

FIG. 12.—Förster's shifting or displacement type of the visual field. (After Wilbrand.)

Förster [109] was the first to examine the fatigue phenomena of the visual field under these circumstances with particular care. His method is as follows: The test-object is carried on the perimeter from the periphery of the *temporal* side through the fixation point to the nasal side along all the meridians, and its points of entrance and exit noted on the chart. It will be found that the visual field obtained has a greater extent in the temporal half,—that is, the side where the test-object first entered the field,—than on the nasal side, because when the latter was tested the patient was already fatigued. If after an interval of rest the central test is made by examining the same meridians in the same order, but now starting from the periphery of the *nasal* side, and the points of entrance and exit of the test-object noted on the chart, it will be found that the visual field is greater in extent on the nasal half because it is the less fatigued half of the field. In other words, two visual fields are obtained, the outlines of which appear to have shifted or been displaced in their relation to each other. For this character of visual field O. Koenig suggested the term "shifting or displacement type," while Schiele described the procedure by the name "diametrical method."[110]

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[109] Loc. cit.

Schiele (quoted by Wilbrand) devised a method of investigating the field of vision to which the name *radial centripetal method* has been given. His investigations were made with a Scherk's perimeter. The meridians were systematically fatigued, only in one-half of the visual field, from the periphery as far as the fixation point, and he found in certain cases that an equally large fatigue defect was found on the same meridians of the homonymous halves of the visual field of the other eye, which had been covered during the previous examination, as on the half of the eye which had been examined first. To his explanation of this phenomenon we shall return later.

III. **Wilbrand's Exhaustion Type of the Visual Field.**—Because Förster's method is time-consuming and very fatiguing to the patient, Wilbrand materially simplified it, confining his examination to the horizontal meridian. Von Reuss, however, declines to consider Wilbrand's method as a simplification of the plan pursued by Förster and prefers to give it a special classification, suggesting the title "Wilbrand's exhaustion type." According to Wilbrand, fatigue phenomena, whenever present, occur on all of the meridians of the corresponding half of the visual field. Hence, in order to determine the defatigability of the nervous system quickly, he proceeds as follows: He uses the horizontal meridian as being the meridian of the visual field of greatest extent. Beginning at the temporal edge of the perimeter, he carries the white test-object along the horizontal meridian with as steady a movement as possible in the direction of the arrow (Fig. 13) toward the nasal half, and has an assistant mark on the diagram with a O the point at which the white test-object enters the field and with a I the point where it disappears. Having reached the point I on the nasal half, the direction of the test-object is at once reversed and it is carried with the same uniform velocity along the horizontal meridian toward the temporal half. The point on the temporal half of the horizontal meridian where the test-object again disappears after its second fatigue excursion is recorded on the diagram by the figure 2. The object is then at once brought back again in the opposite direction and the point at which it disappears on the nasal half indicated by the figure 3, and so on.

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[110] Consult Wilbrand and Koenig, loc. cit.

This method of examination takes little time, and according to Wilbrand, enables the examiner to recognize whether concentric constriction of the visual field was already present at the beginning of the examination, and whether there was with it a slight degree of defatigability; on which half of the visual field this defatigability is chiefly elicited, and whether it can be extended as far as the fixation point or ceases at a definite parallel on the perimeter.

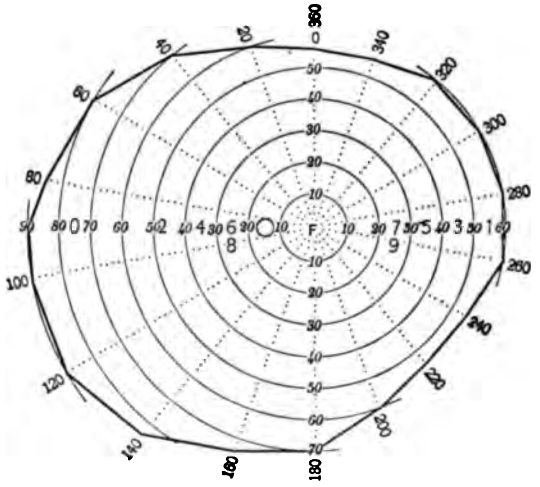

FIG. 13.—Wilbrand's exhaustion type of the visual field. (After Wilbrand.)

To these advantages (quoted from Wilbrand) W. Koenig adds another, namely, that the method affords an accurate means of measuring the patient's attentiveness, or, in other words, his ability to give serviceable answers when examined with the perimeter.

The portion of the visual field which resists fatigue has been called by Wilbrand "minimal visual field." Analogously W. Koenig designates the visual field as it was in the beginning of the "maximal visual field." According to Wilbrand, whenever fatigue is present, it appears on all the meridians of the same half of the field, and the fatigue of the visual field is the same for colors as for white. W. Koenig, however, thinks that while the visual field becomes fatigued for white, it does not lose its reactions to colors at the same time. For example, if a visual field has been fatigued down to the minimal visual field by means of a white test-object, if a colored test-object is brought into the field from the temporal side without any interval of rest, the point of entrance of the said colored test-object approximately coincides with the point where it is first seen after a considerable period of rest. Sometimes, according to Koenig, only a slight difference can be demonstrated, and if no interval of rest is allowed the limits obtained are narrower. The same behavior to white and the remaining colors is observed in the visual field if a colored test-object has been used in producing the fatigue.

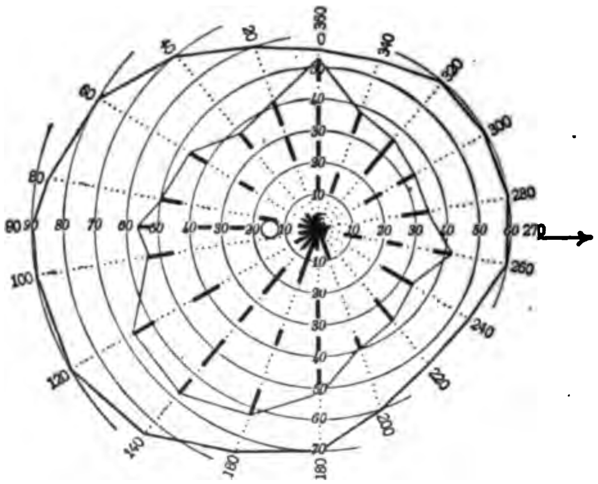

Fig. 11.—Oscillating visual field. (After Wilbrand.)

IV. **The Oscillating Visual Field and Fatigue Scotomas.**—According to Wilbrand, in contrast with types of constant progressive fatigue, cases are occasionally observed in which, to quote his own words, the fatigue pursues, so to speak, a ryhthmical course, the test-object vanishing from time to time along the meridians, to emerge again after a short interval and again disappear and emerge, and so on. For this condition Wilbrand suggests the term *oscillating visual field.* He believes that the condition represents an alternating flaring up and extinguishing of the optic sensory tracts. The same oscillation obtains when a colored object is used. Such visual fields, which are not common, have been observed by him and others, particularly Koenig, in various nervous conditions,—that is, not only in post-epileptic states, but in neurasthenic patients.

These authors and others have also described temporary central scotomas representing fatigue phenomena of the macular portion of the visual field, especially frequent in young persons of neurasthenic tendency. The scotoma may be confined to the fixation point or extend beyond it. It is chiefly manifest to small colored objects, less commonly when the test-object is white. It will be remembered that in the section on hysteria similar phenomena were mentioned, and Von Reuss believes these types of oscillating visual field and scotomas are really hysterical manifestations.

V. **Fatigue Spiral Field (Exhaustion Spiral).**—In ordinary perimetry, the eye being normal, if the points on each meridian at which the test-object is first seen are united with a line, the well-known shape of the visual field appears. If this examination is repeated, an exactly similar map will be produced, or one which differs only slightly from the original. If, on the other hand, according to von Reuss,[111] fatigue of the visual field develops, then, the first field having already been taken, the point at which the test-object in the second trial appears in the visual field on the horizontal meridian, for example, lies nearer to the centre, and this is also the case in all meridians. When the examiner begins for the third time, the point of appearance of the test-object lies still nearer to the point of fixation, and so forth, until further contraction can no longer result and the later points of appearance of the test-objects coincide with the earlier ones. If all of these points are connected, a spiral

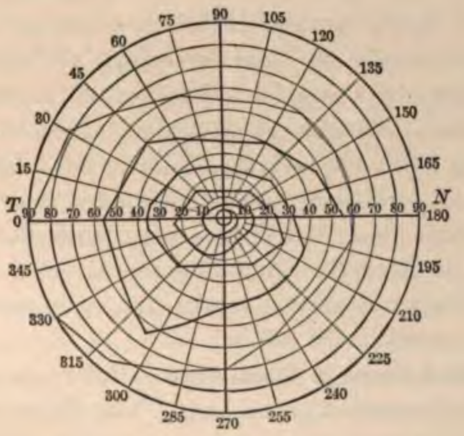

FIG. 15.—Fatigue spiral field. (von Reuss.)

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[111] Loc. cit., p. 64.

symptoms of nervous asthenopia, or by the symptoms which are particularly elicited by mapping the visual field, are often more decided.

Concerning the differential diagnosis from the ocular stand-point of hysteria and neurasthenia enough has been said in the preceding pages and need not again be summarized in the present paragraph, except to reiterate that hysteria presents certain definite ocular stigmata, while those which arise in the course of neurasthenia cannot be said to be peculiar to it, but represent forms of visual fatigue, which may be produced by a variety of nervous affections, although when taken into consideration with other signs, they have their definite place in the symptom-complex of this affection.

As John K. Mitchell and myself [117] have stated, in the difficult distinction between certain types of neurasthenic and hysteric patients, the presence of the disturbance in the color-sense is of some diagnostic import. It is less apt to be present in the former than in the latter, and yet, as we have seen, its absence is of little meaning. It may not be found in many typical cases of hysteria and seems to be present in others which are difficult to eliminate from the neurasthenic category. Von Frankl-Hochwart and Topolanski, who, as before pointed out, believe that neurasthenic patients rarely exhibit concentric contraction of the field, and that their light-sense and even visual acuity are usually normal, have noticed a certain tendency of lowering of color perception, particularly in connection with blue. If this should be substantiated, it might be considered as of some diagnostic importance. Wolffberg's view with reference to colored central color perception has been referred to (pages 683 and 685).

Previously Unclassified Neurasthenic Ocular Phenomena.—Certain systemic writers have described a *neurasthenic conjunctivitis*, which, however, more properly should be regarded as a hyperæmia of the conjunctiva in persons of neurasthenic condition, and which, owing to their nervous instability, even though it be moderate in degree, presents to them a reality far in excess of its real importance. It is probable that this may account for the well-known neurasthenic symptom of foreign body sensation in the eye, sometimes associated with drooping of the lid, to which reference has already been made, a condition which exactly simulates eye-strain and which often is totally unrelieved by the adjustment of suitable glasses, but disappears when with the wearing of these the proper physical and mental hygienic measures are adopted. The same is true of the pricking and burning sensation so frequently complained of by these patients and located along the margins of the lid.

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[117] Journal of Nervous and Mental Diseases, January, 1894.

Ophthalmoscopic phenomena are wanting, or if they are present in the form of various hyperæmic conditions of the eye-ground and congestions of the margins of the optic disc, indicate the signs produced by ametropia.

A rare condition is temporary exophthalmos, particularly when the patient stoops over, which has been described by Oppenheim.

TREATMENT OF THE OCULAR DISORDERS OF HYSTERIA AND NEURASTHENIA.

Naturally, treatment from the general stand-point is indicated, and as this belongs to the domain of the neurologist and general physician, it need not be discussed here, but the eye-treatment of itself is of the greatest importance. First and foremost, there should be the most thorough investigation of the refraction of the eyes, and search made for the smallest degrees of astigmatism. Suitable glasses should be adjusted after the thorough employment of atropine or other suitable cycloplegic, and they should be worn constantly. Anomalies of the ocular muscles require similar careful consideration. Personally, I believe that insufficiency of convergence should be treated by suitable gymnastic exercises with prisms, and I do not believe, a few cases excepted, that the constant wearing of prisms, laterally placed, is a wise procedure. Prisms, base in, associated with reading-glasses, may be required. On the other hand, the symptoms produced by the various types of hyperphoria sometimes yield rapidly if vertical prisms are combined with the correcting lenses, or if a like result is produced by proper decentration. This is not the place to discuss elaborately the propriety of surgical interference. I have often expressed my belief in regard to this matter. I hold that surgical interference is to be considered only when all other measures have been long and faithfully tried and have failed to give relief. While cases of muscular imbalance, best treated by operative interference, are encountered (aptly called by Risley, absolute insufficiencies, equivalent to the structural and insertional anomalies of Duane), in my opinion they represent a limited portion of the whole number. Moreover, as our knowledge of the etiology of the abnormalities of muscular balance increases, our methods of non-surgical treatment improve, and this number grows steadily smaller.

The point, however, should be emphasized, that refractive errors and anomalies of muscular motility are frequently the cause of the entire hysteric and neurasthenic outbreak, and therefore should be among the first problems to be carefully investigated in the management of these cases. This does not mean that ocular treatment should be utilized to the exclusion of general measures. To maintain this would be absurd, but it is equally absurd to attempt cures in many of these cases without consideration of the constantly associated disturbances of refraction, accommodation, and muscular balance. Moreover, it is a distinct mistake to put these patients through long periods of so-called rest-cure, as William Thomson long ago pointed out, without the relief which necessarily must come from the restoration of normality to the eyes. I do not doubt that many a rest-cure would be materially shortened if this plan were adopted. Because a patient lies in bed or rests in his room, there is no reason why he should continue to submit to the plague of eye-strain, which, while it may not be as great as it is in the presence of sustained close eye-work, is constantly in evidence so long as ametropic eyes are utilized in the effort of sight.

This has been fully recognized for years by many neurologists, and accounts for many brilliant results which have been obtained in the modern treatment of functional nervous diseases. Unfortunately eye-strain has been brought into etiological relation with a number of nervous diseases with which it has no such concern by certain injudicious writings, and consequently some physicians have been led to believe that its influence has been exaggerated. This deduction is regrettable and has induced them to neglect the potency of eye-strain in the production and fostering of many neurasthenic and hysteric phenomena.

Because photophobia is a frequent symptom in functional nervous diseases, tinted glasses have been recommended. They should be used with caution, although in some cases excellent results follow the adjustment of lenses which correct the refractive error, and which have been slightly tinted or manufactured of Arundel or lightly smoked glass. Division of the supraorbital nerve to relieve blepharospasm has been recommended (von Graefe, Panas) and practised. It would seem to be an unnecessary procedure and probably acts, as Parinaud says, by suggestion. Indeed, many of these patients readily accept suggestion, and may at times be relieved by this means, hence the success of Christian Science and certain " mind cures." For the same reason magnets have been employed and their use followed by the disappearance of the amblyopia and other signs of ocular hysteria.

THE OCULAR MANIFESTATIONS OF HYPOCHONDRIASIS.

There are no ocular symptoms characteristic of hypochondriasis. On the other hand, some perfectly trivial ocular complaint may be one upon which the morbid fancy of the patient centres; for example, a mild conjunctivitis or a recurring hyperæmia of the conjunctiva may cause him to go from one consulting-room to the other, hoping for relief and believing in all manner of evil consequences. The same is true if he once becomes conscious of the shadows which the formed elements of the vitreous cast upon the retina and to which we give the name muscæ volitantes. They are not caused by hypochondriasis nor are they a symptom in one sense, but they are a condition which may greatly aggravate the mental disturbance. Long-standing hypochondriasis may produce contraction of the visual field, and phenomena which have been described in general terms as fatigue fields, or exhaustion fields, just as they occur in neurasthenic patients.

A matter of some importance is the effect of presbyopia on these patients. It seems to me that there is no doubt, and I find that this is the opinion of some accurate neurologists, that neglected presbyopia or a failure of accommodation, for which no adequate relief is sought, even before the presbyopic age, is frequently the cause of hypochondriasis and mild forms of melancholia. The patients cannot be made to understand that it is the physiological failure of accommodative power, and believe that some form of visual disturbance is arising which later will eventuate in blindness. The irritating inconvenience of oncoming and increasing presbyopia doubtless aggravate these mental conditions under many circumstances. That eye-strain in general may produce hypochondriasis is well known.

OCULAR MANIFESTATIONS OF HYPNOTISM AND SLEEP.

Pansier[118] has devoted a chapter to the ocular conditions during hypnosis and the action of suggestion on the visual function.

During hypnotic sleep the eyelids are usually closed. They can, however, be found partly or sometimes completely open, and the eyes may be rotated upward and the pupils dilated and sluggish.

Although some writers have stated that hypnotic hyperæsthesia may be concerned with all the senses except the sense of vision, this is evidently not an accurate statement, and more than one author has observed increased visual power in the hypnotic state, even when the lids seem closed, because the patient looks through a scarcely perceptible slit in the palpebral fissure. Pansier maintains that there is a hyperæsthetic condition of the retina, which under these circumstances has the effect of rendering it more sensitive to the rays of light. The same author quotes

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[118] Les Manifestations oculaires de l'Hystérie. Oeil Hystérique, Paris, 1892, p. 31.

Binet as having observed in a subject passing from the normal to the hypnotic state a decided augmentation of visual acuity.

Koenigshöfer[119] states that in so far as accommodation is concerned for vision at a distance, there appears to be a relaxation of the ciliary muscle. In effect the hyperope seems to have accentuated his ametropia, the myope, on the contrary, to have diminished it. Now it is an interesting fact first noted by Heidenheim that spasm of accommodation is one of the first signs of beginning hypnosis, and therefore distant vision would be diminished because of the fictitious myopia which is produced. Hence, should there be at a later stage of the hypnosis, relaxation as has just been stated, of the muscle of accommodation, the patient would suddenly find after his return to consciousness, particularly if he was a hysteric, that the visual acuity was suddenly augmented. Pansier quotes Sciamina and Parisotti as having found in their observations a constant augmentation of refraction.

Knies, on the authority of Tamborini and Sepilli, states that the pupil during the cataleptic state of hypnosis is dilated and does not react to light, and quotes Féré as describing dilation of the pupil during cataleptic sleep if the individual is directed to look at a distant object, but contraction of it if he looks at a near object. This has been proved to be the case in a number of other experiments in hypnotism.

According to Koenigshöfer, pupillary reflexes are produced more slowly in the hypnotic than in the normal state. Berger has caused dilation of the pupil after strong sensory irritation in the most pronounced hypnotic sleep.

A number of observers have studied the visual field during the various phases of hypnotism and have found a dilation of the visual field in the somnambulistic stage. Pansier records a number of instances of this character,—for example, a marked restriction of the visual field during waking, while during hypnosis it widened out almost to normal. The visual field for colors in one of his cases was also enlarged, but the colored circles (the patient was a hysteric) preserved their characteristic place and inversion within the limits of the field.

Moravcsik,[120] after pointing out that a contracted visual field may be much widened in hysterical patients by certain irritations,—for example, pouring ether on the hand, touching the tongue with salt, sounding a loud tuning-fork in the ear,—demonstrated in a hypnotic patient that the width of the field may vary according to suggestion. I have studied the visual field during hypnotism, and in one case found it markedly contracted.

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[119] Klin. Monatsbl. f. Augenheilk., xxvi., 1888, p. 13.

[120] Neurolog. Centralbl., 1899, p. 230.

The ophthalmoscopic conditions during hypnotism have been investigated by several observers. In the beginning of hypnotism Förster found the fundus normal. I have had only slight opportunities of making observations under these conditions, but in Weir Mitchell's clinic in the Orthopædic Hospital have not found any alterations of significance, although, as is well known, in the later stages of hypnotism, as particularly has been demonstrated by the researches of Luys and Bacchi, the retinal veins may be much dilated, and this state of passive hyperæmia persisted during the stage of catalepsy. The same phenomena were observed in the phase of fascination. In the cataleptic stage the iris was immobile; in the somnambulistic stage it reacted to light, and at this time the arteries and veins were increased in volume.

In so far as the action of suggestion on the visual function is concerned, it may be stated that all the symptoms of hysteria may be produced by suggestion, and in general terms it may be said that both binocular and monocular blindness has been produced by suggestion, as is especially evidenced by the investigations of Fontan. The dilatation of the visual fields which occurs under the same condition has already been referred to. Koenigshöfer by his experiments confirmed the statement that hypnosis can produce amblyopia and amaurosis and all hallucinations of vision, but the opinion maintained by several authors that color-blindness can thus be produced is, according to him, an error. He maintains that it is not the impression produced by the color which is neutralized, but it is the designation of the color,—that is, the idea of red or green, for example, which is abolished.

As already pointed out, the condition of the pupil and accommodation varies according as the suggestion is made to the subject of viewing objects which are near or far away. This augmentation of accommodation has been proved by Pansier by keratoscopic examinations to occur during suggestion to the subject that he shall look at some object close at hand, while if the suggestion is made that the object is now removed to a long distance, there is relaxation of accommodation and a replacement of the visual axes from convergence to parallelism.

The action of suggestion on the ocular muscles and the artificial reproduction of contractures of the muscles has been studied by Fontan and particularly by Borel, and in this manner ptosis, strabismus, both convergent and divergent, have been brought into existence. Divergent strabismus is much more difficult to produce than the convergent variety of the affection, and it is said that even an immobilization of the eye in the condition of conjugate deviation has been created by similar methods.

The condition of the eyes during ordinary sleep may now receive brief notice. Sleep begins with closure of the lids, or, in other words, with ptosis. This sleep-ptosis Mauthner regarded as representing a temporary nuclear paralysis of the levator. Inasmuch, however, as there is relaxation of the tonus of the sympathetic fibres which pass to the blood-vessels during sleep, Knies and other observers consider that the drooping of the lids is also an evidence of a paresis or, at least, an insufficient action of the sympathetic nerve. This is further confirmed by the fact that during sleep the pupils are contracted, while when the lids are simply closed during waking they are dilated. In other words, together the two symptoms of drooping of the lids and contraction of the pupils would produce the phenomenon, as Knies puts it, of sympathetic ptosis with myosis.

The visual axes are divergent—that is to say, during sleep there is a relaxation of convergence-innervation—and the eyes, to use the language of Hansen-Grut, take up their anatomical position of rest.

Ophthalmoscopic examination of sleeping patients would be extremely difficult to make except under unusual conditions. So far as I am aware, nothing of interest has been developed in this line of examination, although it is possible that there might be some change noted in the calibre of the retinal vessels.

TESTS FOR DETECTING PRETENDED AMBLYOPIA (MALINGERING).

In order to investigate the various amblyopias which occur in hysteric patients it is necessary to apply the tests which are ordinarily used for detecting malingering. To a certain extent they have been referred to in the preceding pages. The subject has been especially investigated by Professor S. Baudry,[121] to whose writings those interested in the great variety of tests which have been devised and which often are extremely useful, especially in medico-legal cases and in work connected with the examination of recruits for the army and navy, should turn. Evidently for practical purposes it is sufficient to briefly describe a few that are in common practice.

The information which may be gained by testing the pupils in their reactions is elsewhere described (see Chapter VI), and we are therefore concerned for the moment with simulated blindness of one eye, simulated blindness of both eyes, and simulated contractions of the visual field.

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[121] Simulated Blindness, System of Diseases of the Eye, edited by Norris and Oliver, vol. iv. p. 801.

Simulated Blindness of One Eye.—To detect this condition the following methods may be used:

(1) **The Diplopia Test.**—This is performed in the usual manner of the ordinary examinations of the external ocular muscles at 5 metres, and a 7 degree prism is placed before the admittedly sound eye. If now superimposed double images are acknowledged, there is binocular vision and the fraud is detected. The examiner may vary the test by placing the prism before the supposed blind eye, either base up or base down.

(2) **Harlan's Test.**—This is an extremely useful and simple device and is performed as follows: Place in position an ordinary trial frame and put before the admittedly sound eye a high convex glass (+ 16D) and before the eye which is claimed to be blind a plane glass or a weak concave spheric (—.25D), which will not interfere with vision. If the proper letters placed at a distance of 5 metres are read, the act of reading must have been done by the eye which was claimed to be sightless, inasmuch as vision at that distance with the other eye is excluded by the presence of the high convex lens. The test may be further elaborated by covering the pretended blind eye and requesting the patient to read the letters. If he is unable to do so the fraud is exposed.

(3) **Baudry's Test with Monocular Diplopia.**—Inasmuch as Graefe's method of producing monocular succeeded by binocular diplopia by means of a prism is rather difficult of application, Baudry has invented an apparatus which consists of a prism supplemented at its base by the juxtaposition of a piece of plane glass equal in thickness to the width of the base; the prism itself is divided near its centre by a section parallel to the base; it thus consists of three pieces, the whole shaped like a piece of the edge of bevelled glass. The apparatus is mounted in a brass cell, covered on both sides. The cover is pierced on each side by a central opening of 6 and 3 millimetres apertures respectively. By a simple mechanism the three pieces of glass slide in the mounting, so that one or other of the two dividing lines with the parts of the glass adjacent may thus be placed before the pupil of the seeing eye,—in other words, either the base of the prism and the piece of plane glass adjacent or the two pieces of the prism itself. Inasmuch as the two dividing lines and adjacent parts look exactly alike, monocular and binocular diplopia can be produced with the greatest ease and without the subject's knowing, even if acquainted with the apparatus, what variety of diplopia (whether binocular or monocular) is present at any given instant.

The method of using the apparatus is simple. The eye that is alleged to be blind is lightly covered by the examiner's hand, and the subject asked to look at the test flame 2 or 3 metres distant and covered with the dark-red glass. The apparatus is then placed before the seeing eye, so as to bring the dividing line between the base of the prism and the piece of plane glass into coincidence with the horizontal diameter of the pupil. The patient sees two lights. The instrument is now removed and adjusted, without his knowledge, so as to bring the dividing line in the prism itself before the central aperture in the metal cell, and placed again before the seeing eye, the other eye being left uncovered, apparently and unintentionally. If the patient now sees double he is convicted, for the diplopia is binocular. If the simulator stubbornly denies the existence of diplopia at first, both phases of the procedure may be repeated in reversed succession.[122]

(4) **Tests with Colored Glasses and Letters.**—These are numerous. The one generally employed, or some modification of it, is known as *Snellen's method.* In general terms the patient is required to look at alternate red and green letters. The admittedly sound eye is now covered with a red glass, and if the green letters are read evidence of fraud is present. Instead of a red glass a green glass may be used, through which the red letters would be invisible.

Very ingenious letters based upon the fact that red upon a white background viewed through a red glass disappears, and viewed through a green glass appears black, have been designed by Dr. von Haselberg.[123] I have found these very useful.

(5) **Tests with Stereoscopes.**—Any of the ordinary stereoscopes may be utilized for this purpose and in the usual manner tests for binocular vision applied. For this purpose Worth's amblyoscope, and especially its modification by Black, should prove useful, on account of the ease with which it is adjusted to suit the position of the eyes and the condition of the ocular muscles. Any refractive error may be previously eliminated by adjusting suitable glasses, which are determined by the shadow-test. With this instrument full investigation of simultaneous vision of dissimilar objects, true fusion and the sense of perspective can be investigated, and its application to pretended blindness of one eye would be very easy.

(6) **Tests by Pseudoscopic Apparatus.**—These depend upon the prop-

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[122] Description taken from T. P. Schneideman's review of Baudry's paper, Ophthalmic Review, xvii., 1898.

[123] Archiv f. Augenheilk., xlviii, 1901.

erties of a plane mirror,—that is, the malingerer is induced to read letters which he believes he is seeing with the admittedly sound eye, when really he is looking at them with the eye which he maintains is blind. An instrument of this character known as the box of Fles is much used abroad, and has consequently been employed in investigating the eyes of hysterical patients. It has been modified in many ways by a number of investigators. In our own country an ingenious test based upon this principle has been designed by Fridenberg, and is known as *Fridenberg's mirror test.*

Tests for Simulated Blindness of Both Eyes.—Naturally, it is much more difficult to detect a malingerer who claims to be blind in both eyes than one who maintains blindness of one eye alone. The tests which have been described are inapplicable, and formerly it used to be stated that the only satisfactory means of detecting the fraud was by placing a careful watch over the patient and taking him unawares. The contraction of the pupil on exposure to light under such circumstances would not prove that there is sight in the eye, because, as Swanzy points out, a lesion of the centre of vision, or in the course of the fibres connecting this centre with the corpora quadrigemina, would produce absolute blindness, and would still permit an imperfect reaction of the pupil to light.

Priestley Smith and Edward Jackson have suggested the following simple test for feigned binocular blindness: Place a lighted candle in front of the subject. Now hold a 6-degree prism base out before one eye. If both eyes see, the one behind the prism will move inward and on removing the prism will move outward, the other eye remaining fixed.

Tests for Simulation of Changes in the Visual Field.—According to Koenig, no case has yet been published in which it has been proved that any one has succeeded in continuously deceiving an experienced physician by simulating concentric restriction of the visual field. Koenig's experiments on this subject are very interesting.[124] He succeeded in simulating a concentric restriction of the visual field without fatigue phenomena to the extent of about 45 to 50 degrees on the temporal side, but when he attempted to simulate a larger visual field the irregularities became great. He was never able to simulate a visual field with only moderate constriction. Similar opinions are held by Wilbrand and Saenger. Simulation of simple concentric constriction of the visual field of high degree may be possible even without preliminary practice with the perimeter, but continuous deception would be difficult.

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[124] Ueber Gesichtsfeld-Ermüdung, 1893, p. 131.

Detection of simulated visual field phenomena could be secured by varying the examination with the campimeter and the perimeter, and especially by altering the point of fixation, as the malingerer would not likely be aware of the changes in the size of the visual field which must take place under these circumstances. In like manner, the tests with different sized test-objects would be sure to bring about detection. Baudry describes Schmidt-Rimpler's method as follows: While the test-object is situated on the limit of the field of vision of one eye, but is still seen by the patient, a prism that is too strong to be overcome is placed before this eye, both eyes being open. It is necessary to ascertain before the adjustment of the prism whether the test-object is seen by both eyes, or by one only. In the former case, if there has been simulation, the patient would have stated that he saw two objects, the true and the false image. If, on the contrary, he had been honest, he would have seen but one object. If originally the object is seen by one eye only, then if the patient is truthful, the prism placed before this eye makes the object disappear; the patient sees nothing. In case of simulation the object remains visible.

Other methods of taking the field of vision will naturally suggest themselves. For example, Koenig's method in which, instead of examining the individual meridians in the usual way, the examiner first picks out one and then another at random, thus rendering it difficult for the subject to remember the same point, as the point where the object enters the visual field. As already noted, von Reuss believes that his fatigue spirals, if present, are a real evidence of the absence of malingering, because they probably would be entirely unknown to the individual who was being examined. The capacity of the eye to recuperate has been recommended as a test for malingering. The principle of the method, according to Arnheim, is that an eye after nervous fatigue requires a greater time for its recovery than one that has become fatigued normally. For example, if a person whose visual field by diffused daylight exhibits a high degree of concentric narrowing very soon presents a normal field in a dark room,—that is, recovers rapidly,—the original statement should be rejected as false. Simulation of the visual field is the subject of a long research by Bichelonne.[125]

Simulation of partial achromatopsia and of dyschromatopsia can best be detected, according to Baudry, by means of Stillings' isochromatic tables, or even better, by those of Pflueger.

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[125] Annales d'Oculistique, cxxix., 1903, p. 252.

CHAPTER XVII.

MIGRAINE, TETANUS, TETANY, EPILEPSY, BLINDNESS DUE TO URÆMIA AND OTHER BLOOD STATES.*

BY JAMES HENDRIE LLOYD, A.M., M.D.

MIGRAINE.

Definition.—Migraine (Megrim, Hemicrania, or Sick-Headache) is a neurosis, characterized by pain in the fifth nerve, by affections of vision, and by gastric irritation. These three groups of symptoms may be said to constitute the tripod upon which the disease rests. In its simple or typical form they are always present, and they appear in a certain order of evolution. Moreover, migraine is a periodical affection, although its periods do not, as a rule, appear with regularity. Its onset is usually sudden; so sudden, in fact, that, like epilepsy, migraine is sometimes called one of the *explosive* neuroses.

As the object of this article is to describe especially the ocular, or ophthalmic, symptoms of migraine, these symptoms will be given special prominence; but at the same time the other classical symptoms of the disease will be stated briefly in order that the account may not lack symmetry.

Varieties of Migraine.—The disease has a variety of forms, some of which have been recognized only in recent years. It is significant that the two chief varieties are named after the ocular symptoms. First of these is the ordinary or classical migraine, which has already been defined, and which is now often called *Ophthalmic* migraine. In this form the affection of vision assumes special prominence; with some well-marked variations in a few cases from the ordinary. Second, *Ophthalmoplegic* migraine, in which a new and distinct element is present, —namely, a paralysis of the third nerve. Third, *Psychical* migraine, in which mental symptoms predominate in place of the usual ocular and neuralgic ones. In addition to these main varieties there are aberrent or abortive types, which, however, can best be described under one or other of the main groups.

Causes.—Migraine has been known and studied for many years. In fact, it is one of the most familiar of nervous diseases, and there is a very abundant literature upon it.[1] In spite of this fact, the essential causation and pathology of the disease are quite unknown, and much that is merely speculative is written about these aspects of it.

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* In the preparation of this chapter the author has to acknowledge the valuable assistance of Dr. C. D. Camp, especially in the literary research.

Sex is probably not a determinative factor of great importance, although some authors, among whom is Eulenburg, contend that migraine is more common in women than in men.[2] But Sinkler has well remarked that this may be due to the fact that men do not seek treatment for the disease as persistently as do women. A far more important factor is *age*. In the great majority of cases the disease makes its appearance before or about puberty.[3] This, in truth, is only the expression of another law,—namely, that migraine is usually an *hereditary* affection. The earlier the disease takes origin the worse and more inveterate it is. Cases in which the onset is delayed until after puberty are not, as a rule, so severe, and some authors have contended that migraine never begins in full adult life. But the statement by Tissot that the disease never begins after the age of 25 years may be open to question. Nevertheless, it is a rule that the longer it is delayed the less severe it is likely to be.

Heredity is the most important factor in the causation of migraine. This law is manifest in a variety of ways. For instance, the heredity may be *direct* or by *transformation*. In the first mode, the transmission is from one or more migrainous ancestors, and may be shown in either a lineal or collateral relation. Thus the patient may acquire the disease from a parent or grandparent, in which case the descent is lineal; or it may appear collaterally, as in an aunt and nephew, or as in the case reported by Chabbert,[4] in which the disease showed itself in two sisters. The descent is said to be by transformation when the patient comes of a neurotic stock, in which other nervous diseases than migraine are prominent in the ancestry, as in Sieffer's case,[5] in which the disease appeared in two children of an insane mother and an alcoholic father. This method of inheritance by transformation is not uncommon in other nervous diseases, as in epilepsy, hysteria, and insanity. When the family stock is neurotic, a nervous disorder may, as it were, be ingrafted upon it instead of being directly inherited.[6] Möbius claims that 90 per cent. of the cases of migraine show heredity, and the disease has been known to descend through four generations, and to show itself in eight members of the same household.[7] Gowers [8] calls attention to, and gives instances of, the association of migraine with other nervous diseases in the same family, especially with epilepsy and pure neuralgia. Its relationship with gout has probably been exaggerated, especially by physicians who practice among an old and gouty population, and see gout in everything, and possibly have it themselves. Trousseau said that "migraine and gout are sisters;" but the kinship is not easily recognized in America.

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[1] Liveing, On Megrim, Sick Headache, and Some Allied Disorders, London, 1873. Eulenburg, Ziemssen's Cyclopædia, vol. xiv. p. 5. Sinkler, Migraine, in Pepper's System of Medicine, vol. v. p. 406.

[2] Eulenburg claims that 5 to 1 is the proportion of women to men; a statement which could only be tested by elaborate statistics.

[3] The present writer, who is a victim of migraine, can recall attacks in the very earliest period of childhood to which his memory extends.

[4] Le Progres Med., 1895, p. 241.

[5] Berl. klin. Woch., 1900, No. 30, p. 657.

There is more reason to suppose that migraine may be related to *gastro-intestinal disorders.* This is in part a popular notion, due to the gastric irritation which is one of the prominent symptoms in many cases, and which is not so much a cause as an effect of the disease. But there is some scientific ground for the belief: attacks may sometimes be aborted by prompt action on the intestinal tract; and the idea is in accord with the opinion that migraine is due to an autoinfection. In some cases as soon as vomiting occurs in the ordinary evolution of that symptom, relief begins, and may even be prompt.

The action of overwork, overstrain, or overstudy may be contributory to attacks by reducing strength, and impairing the resistive power of the system, but further than this it would not be safe or conservative to make statements. The same may be said of shock, moral and physical, as well as of worry, grief, etc.

Symptoms.—Migraine usually begins abruptly. It is, as we have said, one of the explosive neuroses. Some authors describe prodromes, such as a sense of depression or irritability for some hours or even a day beforehand. In rare cases a sense of elation or well-being precedes the attack. But in the commoner type of simple migraine the debut is unannounced. It comes in the midst of work or play, at any time of the day or night; in fact, it is sometimes nocturnal, occurring during sleep, the patient awaking in the midst of a full-blown seizure.

The initial symptom, or *aura,* is usually sensory, and in the vast majority of cases it is visual. Exceptions, however, occur. A sensory aura may occur in other parts of the body, as in a limb. The writer once had a patient who always felt a numb or tingling sensation in his arm. This mounted slowly upward, and as it reached the shoulder, neck, and head, it gradually disappeared, and the pain set in. The analogy with épilepsy is here striking, but an important difference is seen in the fact that the aura of migraine is of much longer duration than that of epilepsy. It is never instantaneous. It may, and usually does, endure for some minutes, even for a half hour.

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[6] Liveing (op. cit., p. 30) discusses Hereditary Transformations. The earliest discussion of this subject was by Moreau de Tours (De l'Etiologie de l'Epilepsie, 1852).

[7] Oppenheim, Diseases of the Nervous System, translated by Mayer, 1900, p. 748.

[8] Diseases of the Nervous System, vol. ii. p. 837.

The patient, of course, from past experience, knows the full import of this aura. He usually drops everything and seeks a retreat, and not seldom he is much disquieted in mind. This mental state during the aura of migraine is important to recognize, for it is at this time in susceptible patients that hysteria may show itself and complicate the clinical picture. We shall refer to this subject later.[9]

The *visual aura* of magraine is characteristic, and has special significance for us in these pages. It is usually a form either of amblyopia or amaurosis. The visual field is obscured in a variety of ways. This is its simplest type. The sight is confused, or partially obliterated. The outlines of objects seen are blurred and indistinct; in other words, there is an amblyopia. The patient is not able to read, or reads with difficulty; the same is true of objects and faces, which he can scarcely recognize. When a true amaurosis occurs, whole sections of the visual field may be obscured, and the patient may even be hemianopic. Thus there may be scotomata, in which large areas, as it were, are blotted out. There may also be scintillating lines, flashes of light and color, blazing or flaming splotches, and zig-zag or whirling figures, the forms of animals, terrifying apparitions, and other curious illusions. In mild cases of ordinary migraine these visual auræ may be but little marked, but in the type of the disease known as ophthalmic migraine they form an important element, and will be described more in detail under their appropriate head.

It is the characteristic of the aura of migraine to disappear before the onset of the pain. This is the almost uniform rule. The visual field entirely clears, so that the patient, if he did not know otherwise from bitter experience, might suppose for a few minutes that he was entirely relieved of his attack. But the respite is brief; in fact, in some cases, the transition from the stage of visual defect to the stage of pain is almost imperceptible.

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[9] Gilles de la Tourette (Traité de l'Hystérie, Paris, 1891) does not mention migraine among the exciting causes of hysteria, an omission which strikes the present writer as rather remarkable.

The pain of migraine is a neuralgia, usually intense, in the distribution of the fifth nerve. It is commonly described as a hemicrania (and in fact the word *migraine* is a French derivative of the Latin *hemicrania*), but it is not always strictly localized on one side of the head. It may be an intense frontal headache, and may even appear to radiate over a large part of the cranium. In other cases it is more distinctly unilateral. Some patients even describe it as limited to one branch of the trigeminal, and the branch most involved is the first or ophthalmic division.

This pain is *cumulative*, as Liveing has pointed out. It begins as a slight affection, but gradually increases, and in the course of an hour or two reaches its maximum. It is aggravated by motion, and by noise and light. The patient usually seeks absolute rest and seclusion. The *character* of the pain is described as boring, or throbbing: Du Bois Reymond, who was a victim of migraine, said that in his own case the pain throbbed in unison with the temporal artery. The *intensity* varies, but it is usually very great; in some cases, however, the pain is mild or even aborts early. Cases have even been described in which it is absent, or replaced by a vertigo; these aberrant types will be referred to again. The *duration* of the pain also varies in different cases, although it is likely to be equal in the same case. As a rule, the pain does not endure over night; it is cured by a night's sleep. Cases in which it lasts for twenty-four hours are rare, although instances are reported in which it persisted for even two or three days.[10] The *termination* of the pain is sometimes abrupt or critical; this is true in cases in which it is relieved promptly by vomiting or by a copious movement of the bowels. It may be, and often is, terminated by sleep.

Gastric disorder is very common in migraine; in fact, it is one of the three characteristic symptoms. It is, as a rule, a late or terminal manifestation. Early in the attack, however, there is complete aversion to food, the patient not only feeling a physical disgust for it, but also apprehending its effect upon the nausea and vomiting which are almost sure to appear in time. That the nausea and vomiting are usually late or terminal symptoms, has long been recognized.[11] In some cases they have the character of a *crisis*, and the pain is often promptly relieved by emesis. This fact gives a hint for treatment. I have known a severe bout of migraine promptly aborted by the administration of an emetic.[12]

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[10] Piorry, Memoirs, etc.

[11] Romberg, "A Manual of Nervous Diseases" (Syd. Soc. Trans., vol. i. p. 176).

[12] An old writer, Robert Whytt (On Nervous Disorders, 1765, p. 305), fully recognized the value of emetics in these periodical headaches. "When they are regular as to their periods, *vomits* should be given an hour and a half beforehand."

The same result can sometimes be obtained by a saline purge. In fact, the vomiting of migraine may be a natural mode of termination; a form of *vis medicatrix naturæ.* It is nature's way of securing relief. In some severe cases, however, the vomiting is not always efficient at once, and it may recur during several hours before complete cure is effected. In some cases the stage of vomiting does not appear; there may be very slight nausea or even none at all. This seems to be so especially of the attacks later in life, when the disease is losing its hold on the patient. But there are distinct aberrent types, even early in life, in which the stage of gastric irritation is conspicuous by its absence.

The vomiting of migraine is often described as a *cerebral* vomiting, especially by the older writers, who affected to see in it an irritation of the pneumogastric nerve. It is true that it occurs without reference to the presence of food in the stomach, just as the vomiting of pregnancy, of sea-sickness, or of meningitis; and in severe cases may afford no relief. It is then attended with retching and great prostration, and instead of relieving, only adds to the patient's suffering.[13]

It is important to bear in mind that the varieties of migraine are not separable by hard and fast lines. They merge, as it were, into one another. Thus the distinction between "ordinary" migraine and ophthalmic migraine as made by some writers is an artificial one. In fact "ordinary" migraine is really only a form of the disease in which the visual auræ are merely not so conspicuous as in the ophthalmic variety. So of ophthalmoplegic migraine; it is similar in many ways to the ordinary type except that it has in addition a motor disorder, —namely, a paralysis of one or other of the ocular nerves. In fact, it might perhaps be justifiable to say that there are two main types of migraine, a *sensory* and a *motor* type. In the one (ophthalmic) a sensory nerve, the optic, is especially involved. In the other (ophthalmoplegic) a motor nerve, the oculomotor, or rarely the abducens, is implicated. We must recall also that there is still another, but rare, type, in which the chief symptoms are *psychical.* These distinctions call to mind the analogy of migraine to epilepsy—an analogy which some authors are particularly fond of pointing out.

OPHTHALMIC MIGRAINE.

Originally the term *ophthalmic* migraine was used to bolster up a theory of the causation of the disease. This theory was that of Piorry, who held that migraine may be caused by overstrain of the iris and overstimulation of the retina. Here we have a very early statement of the theory of *eye-strain,* which has since been worked up to such good purpose by some specialists.[14] The idea, in brief, was that an irritation of the iris and retina was the cause of the disease; hence the theory itself, rather than the disease, should receive the name "ophthalmic." Piorry apparently had no knowledge of the motor, or ophthalmoplegic, form of migraine, and he was merely interested in establishing a plausible explanation of the disease. For him all migraine, however, was not ophthalmic. He recognized that it could be caused by other conditions than eye-strain.

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[13] Liveing, op. cit., p. 136 et infra.

We speak of ophthalmic migraine to-day to distinguish that sensory form in which the visual auræ are conspicuous, as distinct from the motor or ophthalmoplegic form in which there is paralysis of ocular muscles. The credit of first using the term "ophthalmic" for this purely sensory form of migraine is often given to Charcot (as are so many other things in neurology), but as constituting the commonest form of migraine, this variety had really been recognized for many years antedating the great French neurologist.[15] It is true, however, that since Charcot, and probably in part due to his influence, great pains have been taken by many writers to describe minutely the visual disorders of migraine, until now we can almost say that these disorders have been overdescribed.

The visual symptoms in ophthalmic migraine may be divided as follows: 1. Amblyopia; 2. scintillations; 3. scotoma; 4. hallucinations.[16]

By *amblyopia,* as already said, is meant a simple blurring of the visual field. It is perhaps the commonest of these symptoms, and is often seen in simple cases of sick headache. It is not accompanied with distinct scotomata, or blotting out of definite areas of the field, but it is probably closely allied with them. In fact, it appears to be only a minor degree of amaurosis. In this amblyopia the patient sees objects, but sees them indistinctly; the outlines are confused, although with care and attention objects can be made out, as for instance, print or small articles. This would hardly be so if there were distinct scotomata, or localized blind areas. Still, it is difficult sharply to differentiate a mere amblyopia from areas of amaurosis or real blindness. Patients themselves are poor observers of these phenomena; they know that vision is obscured for a time, and they know the import of the symptom, but beyond that they do not attempt to observe or describe. An expert examination in these cases might possibly reveal some small spots of total blindness scattered over the field. But as the symptom is a transient one, lasting only a few minutes, there is little chance to have these cases examined with care. In some cases this amblyopia passes into a stage of scintillation just before it ends.

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[14] Piorry's work was his Mémoire, etc. His theory is discussed in full by Liveing, op. cit., p. 254 et infra. Piorry anticipated many of the specious arguments of later extremists, and, like them, he quoted cases to prove his position. By much the same process of logic it would be possible to prove that eye-strain is responsible for locomotor ataxia, or for cancer of the womb, or, in fact, for any disease which happens to appear in a patient who has a little astigmatism.

[15] Berbez, Les Migraines, in Gaz. Hebdom., 2 S. xxxvi. 19. The disease was recognized by Sazenne (1748), Plenk (1783), Stole (1795), Demours (1818), Wollaston (1824); Madame de Pompadour was said to have been a victim. See also Müller-Syer, Ueber Ophthalm. Migraine, in Berl. Klin. Woch., 1887, xxiv. p. 787.

[16] Galezowski (Etude de la Migraine Opthalmique, in Arch. Gén. de Méd. Juin, 1878) gives a slightly different classification.

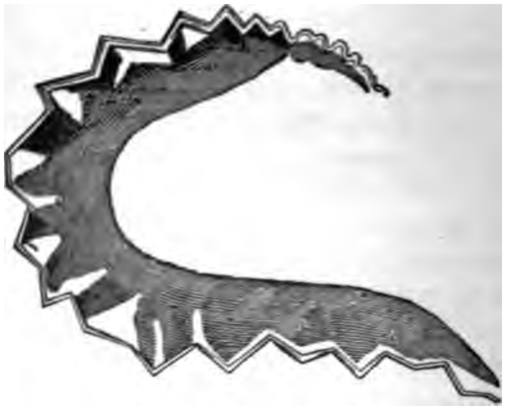

FIG. 1.—Scotoma scintillans. (After Charcot.)

The *scintillations* of migraine are observed in many cases. They consist of waving or vibrating lines of light extending across or at the edges of the visual field. Not unfrequently they are confined to the edges of darkened areas, constituting then the phenomena known as scintillating scotomata. These spectral illusions were fully and graphically described by Liveing.[17] They consist of a luminous border surrounding the blind area. This area, at first small, gradually expands, and as it increases in size, the scintillating rim makes its appearance and grows in intensity. Usually this rim is particolored (although not always so) and it is apt to assume a zig-zag shape, which was described by Fothergill [18] as consisting of luminous angles like those of a fortification. These images have been described in a variety of ways by different observers and they evidently vary somewhat in different cases. They are seen whether the eyes are open or shut, but sometimes better in twilight or darkness. Various colors may be intermingled with them, but in mild cases they are colorless, and not arranged in such fixed patterns as the imagination of some writers suggests. They may, in fact, be mere fleeting and dimly perceived vibrating lines, appearing just before the obscuring of vision passes away. In some cases the scintillation is centrifugal; that is to say, it appears at the edge of the shaded area, and tends to spread towards the circumference of the visual field, gradually dying out. In other, and rarer cases, the scintillation is centripetal, appearing at the edge and gradually extending towards the centre, thus covering the visual field. There has been some discussion whether the scintillations are binocular or monocular, but the weight of opinion is in favor of their appearing in both eyes, a fact which can be proved by the patient closing one eye at a time, when in some cases the scintillations disappear from the closed eye; also as to whether they appear on only one-half of each field (hemiopic), and if so, on which half. We shall return to this subject in discussing the scotoma.

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[17] Op. cit., p. 75 et infra. Liveing gives very graphic colored diagrams of these scintillating scotomata. See the plate at the end of his work.

Besides the wavy and zig-zag scintillations, there are other forms. These have been variously described as showers of sparks, colored hail or rain, flashes of light, flaming bodies, and spots of fire. These may either fill the whole field, or, what perhaps is more common, they appear around the edges of blind areas or scotomata. In fact, where there are genuine scotomata, these pyrotechnics could not well be seen except about the borders of the blind areas. It is possible to suppose that these various illuminations are due to an irritation of the optic fibres; whereas the scotoma, or blind spot, is due to a paralysis of optic fibres. The latter are conceivably the more central fibres, the former are the fibres arranged around the others whose functions are for the time abolished.

These scintillations may be the only visual signs, except some slight amblyopia; but in many cases they are accompaniments of scotomata or blind spots, to be presently described. In some cases they are not seen in the early years of migraine, but make their appearance later in life. Thus in the case of a woman aged 32 years, who had had migraine since childhood, the muscæ volitantes, colored spectra, flashes of light and scintillating scotomata, with photophobia, were only present after the thirtieth year.[19]

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[18] Remarks on Sick Headache, Works, p. 597.

By a *scotoma* is meant a blind spot in the field of vision, and this spot is caused by and corresponds to an area in the retina in which the function of sight is abolished. In migraine such scotomata are not uncommon as part of the visual disorder present in the early stage; in other words, as part of the aura. It is from this symptom that migraine is often called "blind-headache." These scotomata may be variously placed and of variable extent. Thus they may be central, or near the centre, or, as is not unusual, spread over a part of one side of the field. They are, as a rule, present in both eyes. The most important is the scotoma scintillans, already referred to, in which the edge of the blind area is the seat of zig-zag or "fortification" lines of wavy and particolored light.

The effect of these scotomata is to cause curious impairment of vision. Objects are seen only in part. Thus Möbius[20] had a patient who said that men walked about without their heads; and Abernethy, who was a victim of migraine, said humorously that he could see no more of his own name than the "knee" (ne) and the "thigh" (thy).[21] The scotoma lasts but a short time, ten to twenty minutes, rarely as long as an hour; although Galezowski recorded an unusual case in which it lasted for five months, without objective findings. In fact, ophthalmoscopic examination during the time of the attack reveals nothing.

This is the uniform experience. These attacks are sometimes attended with intense photophobia. In some cases, as Möbius points out, the lids droop, but this may be due to an instinctive desire to shut out the light. He also says that pupillary changes sometimes accompany attacks of ophthalmic migraine, but in this he is not confirmed by many observers. They are seen, however, in the ophthalmoplegic form.

These scotomata may be so extensive in some cases as to include one-half of each visual field, in which case there is an hemianopsia. This has been reported by not a few observers.[22] When these scotomata are thus hemianopic they may appear either on the side upon which the pain is present or on the opposite side. When the pain is strictly localized on one side, it is usually on the side of the hemianopsia. This is true also of other sensory auræ, such as a numb feeling in a limb; it is usually on the same side as the pain. Exceptions, however, occur, as was pointed out by Liveing, Calmeil and others. This subject needs more careful study. The hemianopsia may be lateral or vertical.[23]

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[19] Da Costa, "A Case of Ophthalmic Migraine," in the Jour. Nerv. and Ment. Dis., 1890, xv. p. 213.

[20] Migräine, in Nothnagel's Spec. Path. u. Therap., Bd. xii. See also Dianoux, Scotoma Scintillant ou Amaurose Partielle Temporaire. Thèse, 1875.

[21] This must have been a stock joke of Abernethy's, and made to do frequent duty, or else its author has been much misquoted. Abernethy perpetrated this pun in a lecture, published in the Lancet, October 21, 1826, and said the disorder of vision in his own case was caused by an injury (a blow on the nose), and he made no reference whatever to migraine. The lecture has recently been republished in the Lancet (October 22, 1904, p. 1170).

Visual *hallucinations* have been observed in migraine. But they must be rare, for they are reported by only a few observers. S. Weir Mitchell[24] has put such cases on record. Thus a school-teacher, aged 30 years, saw a large, black and hairy dog which disappeared as the pain began. Another patient saw an image of her dead sister. Still another saw an image of a near relative covered with blood, especially if the attack happened at her menstrual period. In a fourth case there was a scotoma scintillans, but *after* some attacks the patient had visions of a trellis of silver covered with vines and flowers of brilliant hue, best seen with the eyes open. Some of these were associated with geometrical figures, pink or red. It would be interesting to know, in such cases, whether there were hysterical symptoms present, or whether such complex visions might be the result of suggestion. Certainly they are not of the ordinary type of visual phenomena seen in migraine, especially when they occur after the attack. The analogy of such cases with epilepsy is also suggested, but of course not proved; for visual hallucinations have been noted among the rare auræ of the sacred disease. A curious form of visual hallucination, or more properly *illusion*, has been noted in migraine by Gowers.[25] This might be described as a projection outward of the scintillation or coruscation; thus to one patient it appeared as though a plate which sat before him on a table was surrounded with the colored spectrum. But neither Gowers nor Liveing mentions true hallucinations.[26]

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[22] Möbius, op. cit. Galezowski, op. cit. Da Costa, op. cit. Dufour, Sur la vision nulle dans l'hemiopie, in Rev. Méd. de la Suisse roun., 1889, p. 445. Schmidt's Jahrb., ccxxvi. p. 74. Liveing, op. cit. Airy, On a Distant Form of Transient Hemiopia, in Philosoph. Trans. for 1870, p. 247. Harris, Hemiopia, etc., in Brain, 1897, vol. xx. p. 308.

[23] Airy made use of the word "teichopsia" (from τεῖχος, a wall, and ὄψις vision) for this symptom, especially where there is the appearance of a "fortification" line.

[24] Neuralgic Headache, with Apparitions of Unusual Character, in Am. Jour. of the Med. Sci., vol. xciv. p. 415.

[25] Diseases of the Nervous System, vol. ii. p. 846.

A few observers have reported cases of that rare symptom known as "hemianopic hallucinations." Harris[27] has collected a few references to these cases, from which it is evident that they are due to organic lesions chiefly. Tuke[28] mentions them in connection with migraine and epilepsy, but Harris gives no instance that was not due to organic disease. The latter author indulges in some speculation about the origin and seat of such visual symptoms, and adopts Hughlings-Jackson's phraseology. For instance, he holds that a visual aura in migraine is caused by an "epileptic discharge"—a needless confusion of the two diseases. In hemianopic hallucinations the patient sees the false images with one-half of the visual fields only.[29]

In rare cases migraine is associated with symptoms of other affections. Among these is *aphasia*. Charcot[30] made the distinction between simple ophthalmic migraine in which the symptoms are scotoma scintillans, transient hemiopia, headache, and vomiting; and *migraine ophthalmique accompagnée*, in which in addition there are a more or less permanent hemianopsia, difficulty in speech, and numbness of one side of the face. He claimed that both the prognosis and treatment are different in this latter and graver type. But this distinction was not original with Charcot, for it had been made long before him by Lebert[31] and others.[32] It is doubtful, however, whether all of the reported cases were true instances of migraine. Some of them appear to have been due to cerebral disturbance, probably vascular or toxic, in which there was not only aphasia but also transient hemiplegia and hemianaesthesia, with mental confusion; and the suspicion is aroused that these attacks in some cases may have been obscure symptoms of Bright's disease, or even of hysteria. Féré[33] also draws attention to the fact that these cases may mean a grave prognosis. He reports a case in a patient, 53 years old, in whom the speech defects became more frequent. There were finally permanent aphasia, right hemiplegia of the face and arm, local convulsions becoming general, and death. There seems to be no sufficient reason to attribute all these grave cerebral symptoms to migraine, simply because they occurred in a migrainous patient. Meige [34] also reports a case, in which there appears to have been disease of the cerebral arteries.

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[26] Zacher (Berlin. klin. Woch., xxix. p. 694) mentions a case in which hallucinations of sight were followed by epileptic attacks, but the case was evidently one of epilepsy with a visual aura, not migraine.

[27] Hemianopia, in Brain, 20, 1897, pp. 318, 360, 361.

[28] Dict. Psych. Med., vol. i. p. 568.

[29] Liveing, op. cit., who described the hemiopia of migraine very fully, apparently makes no mention of hemianopic hallucinations, except the fortification spectra already alluded to in the text.

[30] Leçons de Mardi, 1887, p. 27.

[31] Traité Prat. des Mal. Cancéreuses, Paris, 1851.

[32] Liveing discusses the subject at length in his classical work.

[33] Sur la Migraine Ophthalmique, in Rev. de Med., 1883, iii. p. 194.

Some authors have also spoken of migraine as an early symptom of *general paresis*. It is doubtful what the exact relationship is, even if there be any relationship other than a coincidence.[35] Blocq thinks that the relation is anatomical. He reports three cases, all with neurotic heredity. There is no reason, of course, why a patient with migraine should not also develop general paresis, but a distinct pathological relationship seems not to have been made out. Mickle says that precedent migraine often disappears at the development of general paresis; and Chase [36] observes that severe headache is sometimes seen as an early symptom of paresis, but he does not call such headache migraine. There is possibly some confusion of identity in some of the reported cases.

The same may be said of *locomotor ataxia*, of which migraine is sometimes described as a prodromal affection.[37] This is certainly not observed in the vast majority of cases of tabes, and the association is probably accidental.

That some of the bizarre symptoms noted above are due to *hysteria*, there can not be much doubt. Among these are speech-defects, hemianæsthesia, hemiplegia, convulsions, and other hysterical stigmata. This truth has been fully recognized by some authors, but strangely overlooked by others. In fact, we have in migraine an exciting cause of hysteria. This simple fact is readily understood in view of the alarming nature of the migrainous symptoms, such as the vivid scintillations and blind spots, followed by a period of distressing headache and vomiting. More or less mental or emotional disturbance is not uncommon in the first stage of migraine. The patient is usually, at least, depressed, and sometimes alarmed. He knows full well the import of the visual aura, and understands that he is fated to a painful bout, which will knock him out for a day or two, and entail much physical suffering. It is small cause for wonder, therefore, that in some neurotic patients the symptoms of hysteria should complicate the case. The writer has known such cases, and has been led to suppose that they are more common in children and young persons. Babinski[38] reports such a case in a girl six years old. She had such hysterical symptoms as hemianæsthesia, dyschromatopsia, contraction of the visual fields, and globus; these were associated with, or excited by, the migraine, especially the scotoma scintillans and the violent hemicrania. To show how susceptible such patients may be to suggestion, it was only needful to make pressure on the sixth dorsal vertebra (in this case, a hysterogenous point) to cause the appearance of a scotoma. In some cases in which so-called epileptic equivalents have been reported, the convulsions were probably hysterical.[39] In cases in which the scotoma is reported as unduly prolonged (as in Galezowski's case, referred to on a preceding page) it is likely that the symptom is due to the great neurosis.

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[34] Rev. Neurologique, No. 18, 1904.

[35] Parinaud, Migraine Ophthalmique au debut de la Paralysie Générale, in Arch. de Neurolog., 1883, v. p. 57. Blocq, Migraine Ophthalmique et Paralysie Générale, in Arch. de Neurolog., xviii. p. 321.

[36] General Paresis, 1902, p. 146.

[37] Raullet, Etude de la Migraine Ophthalmique, Thèse de Paris, 1883.

Reference has already been made to the forced resemblance between migraine and *epilepsy*. Both diseases are explosive, both are preceded by an aura, and both occur in neurotic patients, but beyond these few points the resemblance ceases, or at least is not marked.[40] An important question arises, however, as to the possibility of the transition from migraine to epilepsy. This claim is made by some authors, but the present writer has never seen or heard of an authentic case, and does not believe in the doctrine. The cases reported are probably instances of hysterical convulsions, or they are cases in which migraine and epilepsy coexist in the same patient—which is by no means a proof that the one disease causes or merges into the other.[41]

Pathology.—It is proper here to make a brief reference to the claim that migraine is caused by *eye-strain*. As already said (page 703), this theory was advanced by Piorry many years ago, but it fell into obscurity until revived recently by a few extremists. Martin has reported no fewer than 352 cases in which he thought he was able to detect a low degree of astigmatism as the cause of migraine,[42] but he probably mistook a *post hoc* for a *propter hoc*, a conceivable error in a hasty observer in view of the wide prevalence of astigmatism. Gould,[43] who has written voluminously on the subject, makes the sweeping claim that the near use of astigmatic eyes is always required to produce migraine, but his conclusions are widely rejected. Paul Bert[44] held that scintillating scotoma, generally attributed to fatigue of the retina, is in reality due to fatigue of the nervous centres; but there seems to be no real scientific warrant for such dogmatic statements.

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[38] De la Migraine Ophthalmique Hystérique, in Arch. de Neurolog., November, 1890, p. 305. In this paper Babinski reports several cases of hysteria caused by migraine. Among the hysterical stigmata were mutism, hemianæsthesia, anæsthesia of the palate, monocular diplopia, and contraction of the visual fields. He even claimed that attacks had been cured with hypnotism. Thomas (Migraine Ophthalmique Associée de Nature Hystérique chez l'enfant, in Rev. Med. de la Suisse, xii. p. 800) reports the case of a boy whose migrainous attacks were accompanied with paresis of the right side and difficulty in speech, lasting an hour, with other stigmata of hysteria. See also Fink, Des Rapports de la Migraine Ophthalmique avec l'Hystérie, Paris, 1891.

[39] Mengazzini, Rev. Sper. di Freniatria, abstracted in Cent. f. Nervenheil., xvii. p. 162.

[40] Möbius, Ueber Migräne, in Cent. f, Neurolog., viii. p. 244.

[41] Spiller, The Relation of Migraine to Epilepsy, in Am. Journ. Med. Sci., 1900.

Attempts to describe the pathology of migraine have been equally speculative and futile. Dufour[45] held that the disease, or rather the scotoma, was due to "vasomotor anæmia" of the occipital region of the brain. Ball[46] supposed it was due to a "local asphyxia." Latham[47] believed that the visual disturbance was due to "excitation of the sympathetic," and so on. Recently the tendency has been to suppose migraine to be due to an infection; but this theory is merely an evidence of the present trend towards a humoral pathology, and it cannot be said to rest upon solid facts.

German writers, as Eulenberg,[48] have been fond of insisting on the distinction between the "angiospastic" and "angioparalytic" types of migraine. The distinction is based on the supposed condition of the vasomotor supply. In one form the face is pale, the eye hollow, the pupil dilated, and the arteries tense. Eulenberg even claimed to have found the temperature of the external ear reduced four-tenths of a degree! In the other form the reverse of all these conditions is seen, and the temperature of the ear rises *two-tenths* of a degree. These fine physiological studies have never thrown the slightest light on the nature of migraine, for the question still remains, What causes the vasomotor change?

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[42] Ann. d'Oc., xcix. pp. 24 and 205.

[43] Journ. Am. Med. Asso., Jan. 1904.

[44] Comp. Rend. des Séances de la Soc. de Biol., 1882, p. 571. The very extravagance of the claims of some of these writers tends to cause distrust of their opinions.

[45] Sur la Vision Mille dans l'Hemiopie, in Rev. Méd. de la Suisse, 1889, p. 445.

[46] Quoted by Raullet, Etude de la Migraine Ophthalmique, Thèse de Paris, 1883.

[47] On Nervous or Sick Headache, 1873.

[48] Op. cit. Quaglino (Annali del Ottamologia, 1871, p. 7, cited by Raullet) held that ophthalmic migraine was due to "spasm of the arteries of the retina." It is extraordinary how completely the advocates of these vasomotor theories ignored the question of a possible cause for the alleged action of the vasomotors. There could hardly be a vasomotor spasm without something to cause it. These theories grew out of the *solidist* school of pathologists, who explained every disease by a change in the tissues, and complacently ignored the necessity for finding causes for such alleged changes.

Among other works on ophthalmic migraine are the following: Robiolis, Contribution a l'Etude de la Migraine, etc., Thèse de Montpellier, 1884. Baralt, Contribution a l'Etude du Scotome Scintillant, etc., Thèse de Paris, 1880. Dianoux, Du Scotome Scintillant, Thèse de Paris, 1875. See also Brewster, Philosophical Magazine, 1865, p. 503.

Some pathologists reach impotent conclusions about migraine, as, for instance, Oppenheim, who, at the autopsy of an old migrainous patient, found central softening from embolism as the cause of death, and who thought vasomotor disturbance had caused the old migraine and likewise the recent embolus. Surely this is a far-fetched conclusion.

OPHTHALMOPLEGIC MIGRAINE.

This form of migraine is characterized, as its name implies, by a paralysis of some of the ocular muscles. This paralysis is usually a sequel or result of the migrainous attack, and is, of course, like that attack, periodical. The muscles involved are, as a rule, those supplied by the third nerve, but a few cases have been observed in which the paralysis has occurred in the fourth or the sixth nerve.

The term "ophthalmoplegic migraine" was suggested by Charcot[49] in 1890, but Charcot was not the first to record an instance of this interesting disease. Möbius[50] is usually credited with this distinction (in 1884), but even before that date Adams,[51] in 1877, had reported a case of paralysis of the left third nerve with remarkable intermission, relapse, and recovery, which may have been an instance of ophthalmoplegic migraine. Saunby,[52] in 1882, had reported a clear case of migraine with paralysis of the third nerve, and the distinction of priority seems indubitably to belong to him. After 1884 reports of cases became quite numerous; and merely to repeat the titles of the papers would now be a task quite irksome and prolonged.[53]

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[49] Sur un cas de Migraine Ophthalmoplegique, in Cliniques des Maladies du Syst. Nerv., 1892, p. 70.

[50] Neurol. Beitrag., iv. Deut. Zeit. f. Nervenheilk., Bd. xvii. 299.

[51] Ophthalmic Hosp. Rep., Dec., 1877.

[52] Lancet, 1882, Sept. 2, p. 345.

[53] The following are the principal early reports besides those already named: Snell, Lancet, 1885, vol. ii. p. 939. Clark, ibid. Beevor, ibid. Thompson, Char. Ann., 1885, p. 567. Manz, Berl. klin. Woch., 1885, Nr. 40. Parinaud, Soc. d'Oph. Francaise, Janv. 29, 1895; also Schmidt's Jahrb., Bd. ccvii. p. 245. Suckling, Brain, 1887, July, p. 241. Senator, Zeit. f. klin. Med., Bd. xiii. p. 252. See Charcot's paper, op. cit., for a brief discussion of the history, with references to early cases.

In ophthalmoplegic migraine we have that *motor* type of the disease, to which reference has already been made, in distinction from the *sensory* or ophthalmic type, which has just been described. This distinction seems warranted by the fact, as pointed out by Charcot and others, that the ophthalmic aura, such as the scintillating scotoma, is often absent in this motor form, whereas the paralysis of some of the ocular muscles is pronounced and characteristic.

Ophthalmoplegic migraine generally pursues about the following course: The attack is usually divided into two periods. First, the period of pain. Second, the period of paralysis.

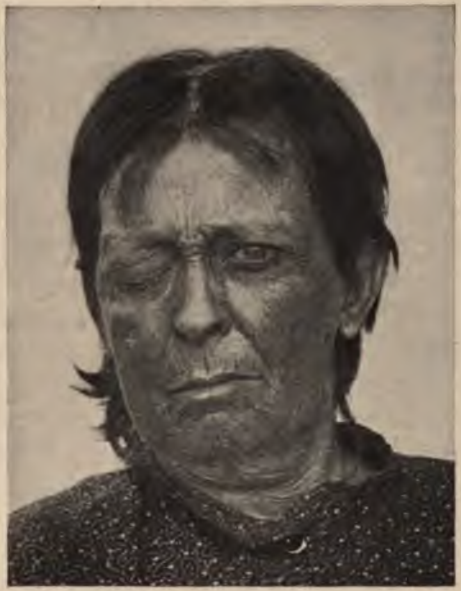

FIG. 2.—Paralysis of the right third nerve, to illustrate ophthalmoplegic migraine, although in this case the disease was brain syphilis.

The *period of pain* begins brusquely, and the pain itself is localized upon one side. This localization is more marked than in ordinary migraine. The pain is often centred about the eye,—*i.e.*, in the ophthalmic division of the fifth nerve. The visual aura, such as scotoma scintillans, hemianopsia, etc., is usually absent.[54] Although the pain begins suddenly, it does not reach its maximum at once, but gradually increases in severity; and although it is usually localized, it may in some cases extend even to the occiput. But it almost invariably remains upon one side. This pain may become most severe. Its duration varies in different cases; in some it is short, a matter of a few hours; in others long, extending over some days. As in ordinary migraine the bout of pain is usually terminated by vomiting. This vomiting may be in the nature of a crisis: it appears to bring relief, and this relief is prompt, even sudden. In other words, the termination of the painful period is as abrupt as its beginning.

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[54] Charcot, op. cit. Suckling, op. cit. Kollarits, Deut. Zeit. f. Nervenheilk., 1904, Bd. xxvi. H. 1, 2.

As the pain and vomiting disappear the second period, or *period of paralysis*, asserts itself. This paralysis has certain characteristics, which are quite common in the majority of cases, although exceptions to the rule occur, as will be noted. The palsy is an ophthalmoplegia, and in the great majority of cases it is confined to the third nerve. The paralysis of the third nerve is usually *total* and *complete*. That is to say, it is total in the sense that all the branches of the nerve are involved, and it is complete in the sense that the muscles are not merely weakened but absolutely paralyzed. Hence we have presented the classical picture of a third-nerve palsy, such as occurs from an organic lesion. The upper eyelid droops, the eyeball is rolled outwards, and the pupil is dilated. Moreover, the pupil is quite immobile; it does not react to light, nor upon convergence or accommodation. Thus there is an ophthalmoplegia-both *externa* and *interna*.

The *duration* of these two stages varies, as we have said, in different cases. The period of pain is usually shorter than the period of paralysis. This paralysis, indeed, may last for a comparatively long time. In Schilling's case [55] it endured for from four to six weeks; in Parinaud's case for from two to three months. But in many cases the paralysis disappears in a few days.[56]

Charcot claimed that the paralysis is always *confined to one eye;* it never shifts from one eye to the other, and in a given case it always appears in the same eye in succeeding attacks. It may be in the left eye or in the right eye, but always in the same eye in the same patient. But this rule has been found by other observers not to be constant. Thus Demicheri [57] observed a case in a woman aged 64 years in which the palsy appeared first on the left and then on the right side; and Karplus [58] had a case in a woman aged 39 years in which there was a left-sided oculo-motor paralysis following migraine, which reappeared on the right side. These cases, however, appear to be quite exceptional.

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[55] Münch. med. Woch., 1903, No. 18, p. 776. See also Wilbrand and Saenger, Die Neurologie des Auges, I, 2 Ab., p. 575.

[56] Manz, op. cit.: Berthen, Klin. Monatsblat. f. Augenheilk., Sept. 1893.

[57] La Clinique Ophthalm., 1899, 18.

[58] Jahr. f. Psych., xii, p. 158.

Some cases of this recurrent palsy of the oculo-motor nerve observe a distinct *periodicity*, but this is not true of all. Kollarits's patient had attacks three or four times a year. In Suckling's case they occurred every two weeks; in one of Karplus's patients every year. In women the attacks have occasionally been seen to coincide with the menses. On the other hand the occurrence has sometimes been most irregular and at very long intervals. In one case [59] the onset was in the patient's thirteenth year, and the attack was very brief (only one day); then after an interval of ten years the disease recurred, and later became periodical at short intervals—two or three attacks monthly. Charcot called special attention to the fact that the duration of the access of ophthalmoplegic migraine, even the shortest, is usually longer than the access in ordinary migraine; and he thought it was the rule that in cases in which the attacks were short the intervals between the attacks were also short. When the attack does not exceed four or six days, the seizures may occur as often as once a month, as in Senator's case.

The *onset* of migraine in these cases is often at a very early age; in fact, the majority of them seem to date their origin from childhood or even from infancy. Thus in one case [60] the first attack happened when the patient was only a year old; the second attack occurred at seven, and thereafter the attacks recurred every nine or ten months. In Suckling's case the attacks had occurred also since infancy; and de Schweinitz [61] has reported a case which began in an infant one year and a half old, and which, after repeated attacks, left a permanent paralysis of the third nerve. Not a few of the cases began before or at the age of puberty; thus cases are recorded which had their onset at 5½, 7, 8, 11, 13, and 14 years.

It is a noteworthy fact that there is often a *change of type* in these cases. Thus in the early years the disease presents the type of an ordinary or ophthalmic migraine, and this changes later to the ophthalmoplegic type. This seems to prove that the latter form is a genuine migraine, and not a different disease, as some observers have claimed. This fact, however, does not avoid the inference that some few exceptional cases of recurrent palsy of the third nerve are not cases of true migraine, a fact which will be discussed under the head of diagnosis. In cases, however, in which there is this change of type the correlated facts are too obvious to admit of a doubt that the two types are really the expression of one and the same morbid entity. Chabbert[62] reports the case of two sisters who had ophthalmic migraine with scotoma scintillans, hemianopsia, etc. In one of these cases at 52 years the attacks became complicated with right-sided oculomotor palsy. Joachim[63] had a patient who had ordinary migraine from the eleventh year; at twenty years of age she suddenly developed oculomotor palsy with diplopia, etc., after a rather slight attack. Somewhat similar cases have been reported by Stezeininski,[64] Bouchaud,[65] Stock,[66] Senator,[67] and Lapersonne.[68] In Bouchaud's case the oculomotor palsy developed in a woman aged sixty years who earlier in life had had migraine. Charcot[69] records a case in which ordinary migraine had existed in early life and had ceased in the patient's twenty-third year; but in her thirtieth year she began with attacks of recurring oculomotor palsy. It is impossible, as Charcot says, not to see some relationship between these two types, even widely separated as they were in time.

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[59] Darkschewitsch, Arch. f. klin. Med., Bd. xlix. S. 457.

[60] Ormerod and Holmes Spicer, A Case of Recurrent Paralysis of the Third Nerve, with Migraine, in Lancet, Dec. 21, 1895, p. 1580.

[61] Recurrent Oculomotor Palsy, with a Case, in Boston Med. and Surg. Journ., 1895, p. 341.

It has been observed in some cases that after repeated attacks of ophthalmoplegic migraine the paralysis tends to become more and more persistent and even in time to become *permanent*. This is true especially of those cases in which the period of paralysis is prolonged and occurs at prolonged intervals. In such cases the intervals between the attacks are not always entirely free from the appearance of some paresis; and even the pain may persist in slight degree. Senator, noting these facts, said that the disease was not so much periodical as it was "continuous with periodical exacerbations." The suggestion of an organic lesion, such as an inflammation of the nerve-trunk, is likely to occur to the observer of these cases; and in fact some such organic lesion may be the cause of the permanent palsy. In Schilling's case there was some interparoxysmal paresis; as also in Ballet's case.[70] In a case recorded by Karplus[71] a permanent ptosis was left; an interesting instance of only one branch of the third nerve being permanently injured. In de Schweinitz's case in a child a divergent squint remained in the intervals, but the ptosis appeared and disappeared with each succeeding attack until the twenty-eighth year, when the ptosis too became permanent. In Ormerod and Spicer's case some of the paralyzed muscles had never recovered. Whatever may be the explanation of this permanent palsy in a disease which is usually called "functional," it is necessary for the present to hold in abeyance any theory which does not include the possibility of an organic lesion. It is likely indeed that this permanent injury to the motor nerves is the result of some irritant, such as a toxin, acting as the essential cause of migraine.

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[62] Le Progrès Méd., 1895, No. 15, p. 241.
[63] Deut. Arch. f. klin. Med., Bd. xliv. p. 185.
[64] Rec. d'Ophthalmol., September, 1897.
[65] Presse Méd., Avril 28, 1897.
[66] Cited by Paderstein, loc. cit.
[67] Loc. cit.
[68] Progrès Méd., 1903, No. 10, p. 161.
[69] Op. cit.
[70] De la Migraine Ophthalmoplegique, Thèse de Paris, 1896.
[71] Jahr. f. Psych., vol. xii. p. 158.

The paralysis in ophthalmoplegic migraine is not always limited to the third nerve, and in a few rare cases the third nerve has escaped entirely, while *the fourth* or the *sixth nerve* has been implicated. Leizenberger[72] reported the case of a man aged 29 years who had migrainous attacks every eight days followed by isolated paralysis of the trochlear nerve. Ordinarily when the fourth nerve is involved, it is in association with the third nerve, as in the case recorded by Coutouzis,[73] in which there was conjoint palsy of the oculomotor and trochlear nerves with violent headache. The attacks were distinctly migrainous, with pain in the parietal region, vomiting and salivation for forty-eight hours, followed by the loss of power in the two nerves. There was no scotoma or hemianopsia. The paralysis lasted for twelve days, but longer in the superior rectus muscle.

Paralysis of the *sixth nerve* has been observed in a small number of cases. Charcot has made such a case the subject of a detailed report.[74] The patient, aged 41 years, had ordinary migraine beginning at the age of 16, which ceased at 32 years, to be followed at 38 years by a violent attack of left-sided neuralgia without nausea or vomiting, and lasting for four hours. Diplopia resulted, and was due to an isolated paralysis of the abducens nerve, lasting for five days. After an interval of two years a similar attack, but on the *right* side, occurred, and was followed by paralysis of the oculo-motor nerve; six months later occurred still another attack, but on the *left* side, followed by paralysis again of the sixth nerve. Charcot was obliged to conclude from this case that the attacks are not always unilateral; the fact was shown, however, that the third and sixth nerves, although paralyzed in opposite eyes, were paralyzed each on the same side in each attack. This palsy in

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[72] Abstracted in Neur. Cent., xvii., 2, p. 73.

[73] Journ. de Méd. et de Chir. Prat., Avril 25, 1897.

[74] Rev. Neurolog., 5, 8.

Charcot's case was long continued, but not in all branches equally, as the levator palpebræ and superior rectus were most, or longest, involved. Pulerstein[75] reported a case in which there was a paralysis of the left abducens nerve and a weakness of the left superior rectus muscle, with equal pupils and no paresis of the levator palpebræ; showing again that the various branches of the third nerve are not always equally at fault. De Schweinitz[76] also put on record a case of recurrent palsy of the abducens nerve, which began when the patient was only one year old, and recurred one year later, and again in five years. The attack was ushered in with fever, nausea, vomiting and headache, and recovery after each attack was complete.

A very rare case was that reported by Rossolimo,[77] in which a recurrent paralysis of the *seventh* nerve was always preceded by a migrainous attack. The paralysis was in the superior as well as the inferior distribution of the nerve; so it was evidently peripheral. There was no involvement of ocular muscles. Rossolimo referred to a similar case reported by Hatschek.[78]

It is a remarkable fact that but few authors make mention of anæsthesia of the *fifth nerve* in these cases of ophthalmoplegic migraine. The inference is that such anæsthesia seldom occurs. When we consider the fact that the nerve-storm vents a large part of its fury on the fifth nerve, causing the intense headache, and that the cause, whatever it is, is sufficient to paralyze motor nerves, it seems rather remarkable that the sensory nerve is not also paralyzed. Knapp[79] has reported a case in a man aged 11 years in which there was a distinct anæsthesia in the first and second divisions of the fifth nerve. The attack was of long duration, and the sensory condition was what is known as anæsthesia dolorosa; that is, there was a partial loss of sensation, the face feeling as though it were covered with leather, and objects were felt as through this substance; associated with pain on pressure or even on slight touch. Haynes[80] also has reported a case in which the eye felt sore, and there was anæsthesia of the supra-orbital branch of the fifth nerve, accompanied with or followed by ptosis.[81]

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[75] Deut. Zeit. f. Nervenheilk., xv., 5 u. 6, p. 418.

[76] Philada. Polyclinic, vi. 39.

[77] Neur. Centbl., 1901, S. 744.

[78] 1894, Klin. von Nothnagel. See also Knapp's paper, cited below.

[79] Boston Med. and Surg. Jour., September 27, 1894, p. 308. Knapp gives a bibliography including many of the reported cases of recurrent oculomotor palsy up to that date.

[80] Recurrent Ptosis, in N. Y. Med. Journ., 1897, No. 7.

[81] See the case, reported by the present writer, of unilateral complete and total

Some authors speak of recurrent oculo-motor palsy *without pain,* and cases have been reported, but they are probably not migrainous.[82] In fact there is not a little chance of confusing cases of organic paralysis of the third nerve with cases of migraine. This subject will be referred to more at length presently.

Troemner[83] has reported a rare case, namely, ophthalmoplegia *interna* following severe attacks of migraine. There was no palsy of the extraocular muscles. The right pupil was widely dilated and immobile to light.[84]

Diagnosis.—Paralysis of the oculo-motor nerve is not an uncommon result of organic diseases of the brain, among the most common of which are brain-tumors and brain-syphilis. Both these lesions are productive of pain, and in both of them the palsy of the third nerve may be paroxysmal, although this is less likely to be so with tumors than with syphilis. The latter affection is indeed very apt to cause oculomotor palsy. It was a favorite saying with Ricord that syphilis puts its sign manual on the third nerve. Thus an error in diagnosis might easily be made in cases which at first view may appear to be migrainous.

In order to establish the differential diagnosis the history and course of the case must be considered. In cases of brain-tumor the symptoms are not usually limited to the third nerve; there are other evidences of intracranial disease, such as choked disk, possibly convulsions, and other and more widespread paralyses.[85] Moreover the symptoms do not intermit so markedly as in migraine; the course is steadily progressive.[86]

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ophthalmoplegia, with anæsthesia of the fifth nerve and optic atrophy, in a woman with syphilis. (Univ. Med. Mag., October, 1899.)

It is well also to be on guard against a mere *hysterical* anæsthesia in the territory of the fifth nerve. The present writer has reported (Phila. Med. Journ., March 30, 1901) a case of Bell's palsy in which the whole side of the face and head was the seat of an hysterical anæsthesia. It is to be noted in such cases that the anæsthesia is not confined strictly within the limits of the fifth nerve, for it is a peculiarity of hysterical anæsthesia everywhere not to be located within the exact distribution of any particular nerve.

[82] Schmidt-Rimpler, Handbuches von Nothnagel, Bd. xxi.

[83] Cent. f. Nervenheilk., October, 1899.

[84] The following papers on the general subject of ophthalmoplegic migraine have not been obtainable: Marina, Ueber Multiple Augenmuskellähmung, Wien, 1896; Chiarini, La Emicrania Optalmoplegica, Rif. Med., 1895, xi. 169; Graef, Mauth Vortrag. xiii. 398.

[85] Richter (Arch. f. Psych., 1887, Bd. ix. p. 259) records a case of recurrent palsy of the third nerve caused by a fibrochondroma of the dura involving the nerve. There were epileptic attacks, and the case differed otherwise from the syndrome of ophthalmoplegic migraine. See also Karplus (Wien. klin. Woch., 1895, No. 50).

[86] For a case of brain-tumor causing paralysis of the third nerve see a report by the present writer (A Case of Tumor of the Mid-Brain and Left Optic Thalamus,

In the case of brain-syphilis the difficulty of making a diagnosis may be greater, and in fact some of the reported cases of recurrent oculo-motor palsy have probably been of syphilitic origin. The history of many preceding attacks, always the same, would be in favor of migraine, as would also the transitory nature of the attack in most cases. Optic neuritis may or may not be present in brain-syphilis; it is never seen in ophthalmoplegic migraine. In case the history is obscure great care should be exercised in making this diagnosis.[87]

The nuclear ophthalmoplegia, described by Hutchinson, is an insidious and progressive disease in which as a rule one nucleus, and therefore one nerve branch, after another is involved, and in which there is not a tendency to periodical recurrence and recovery. Moreover the affection is usually bilateral, and not accompanied with pain, although in the case reported by the present writer pain was a symptom.

A history of syphilis can be elicited in some of these cases of ophthalmoplegic migraine,[88] but that is not necessarily a proof that this affection is syphilitic. In the great majority of cases there is no such history recorded.

A basilar pachymeningitis has been known to cause a one-sided oculomotor palsy with symptoms resembling migraine.[89] The same has been observed in a case of tubercular meningitis by Weiss,[90] in whose case there was tuberculosis of both lungs, and a recurrent palsy, the last time permanent, of the left third nerve. Wadsworth [91] records a case associated with otorrhea, but the association was probably a mere coin-

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in the Med. News, January 30, 1892, p. 129). In this case there was a *crossed* paralysis,—*i.e.*, the third nerve on the side of the lesion and the arm and leg on the opposite side were involved. This crossed paralysis, due to a lesion in the mesencephalon, has been called by the French the syndrome of Weber. It is significant of an organic lesion, although it may be closely simulated by hysteria.

[87] The present writer has recorded three cases of ophthalmoplegia of various kinds, all of organic origin. The first was a case of total and complete unilateral ophthalmoplegia (interna and externa) caused by a syphilitic neoplasm at the base of the brain just behind the orbit. (Annals of Ophthalmology, January, 1898.) The second was a case of nuclear ophthalmoplegia in a woman, closely following the type described by Hutchinson. In this case the palsy was not unilateral, neither was it total; that is to say, not all the orbital muscles were involved. (Philada. Med. Journ., June 3, 1899.) The third case was one of unilateral ophthalmoplegia, total and complete, associated with anæsthesia in the fifth nerve and atrophy of the optic nerve—probably caused by syphilis. (University Med. Mag., October, 1899.)

[88] Mengazzini, cited by Möbius, records a syphilitic case. See also Leaffer, Berl. klin. Woch., 1900, No. 30, p. 657.

[89] Gubler (Dis. Inaug., Dresden, 1897). See also Ziehen (Neurol. Centblt., 1899, xviii. 4, p. 173). Also de Schweinitz's case (Philada. Polyclinic, vi. 39).

[90] Wien. med Woch., 1885, 17.

[91] Trans. Am. Ophthal. Soc., 1887, iv. 460.

cidence. The periodicity of some of these cases has naturally suggested malaria as a cause,[92] but the relationship is not clear; although the present writer has seen a case of transient paralysis of some of the ocular muscles in a well-marked case of malarial poisoning in his service at the Methodist Hospital. Remak [93] has recorded a case of ocular palsy in a case of juvenile muscular atrophy, but it has no bearing on the subject of migraine. Thompson [94] had a case associated with convulsions and dementia in a child aged 5 years, and one of Joachim's cases was in a backward child who also had epileptiform convulsions.[95]

In a few cases of recurrent oculo-motor palsy there has been noted a nasal catarrh. This was seen in Knapp's case; also in a case recorded by Ahlstrom.[96] The latter observer thought the catarrh was the cause of the palsy, which he believed was "reflex." But the query arises whether the catarrh may not be an *effect* of the involvement of the fifth nerve supply to the mucous membrane of the nose. The symptom has not been recorded in many cases. Möbius [97] and Bernhardt [98] suggested an "autoinfection" as the cause, or it may be "rheumatic." These ideas are merely speculative.

In a case recorded by Karplus in which the attacks occurred as a rule every two or three months, no seizures occurred during pregnancy. This exemption is analogous to what is seen in epilepsy in not a few cases.

Some observers have seen a relationship between tabes dorsalis and migraine; a relationship which is suggested especially by recurrent palsy of the third nerve in locomotor ataxia. Thus Oppenheim [99] in 85 cases of tabes found 12 cases with attacks of migraine, only two of which, however, were of the ophthalmoplegic type. He claimed that migraine is a frequent symptom of beginning tabes. But it seems of doubtful propriety to call the recurring palsies of the third nerve in tabes instances of migraine. They are due to the pathological process underlying the tabes, and the history and clinical course are not the same as in migraine. It is desirable, moreover, to avoid mistakes, such as overlooking the onset of locomotor ataxia, and calling a tabetic third-nerve palsy a symptom merely of migraine. The same may be

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[92] Klialschkin, Neurolog. Centblt., 1897, xvi., 5.

[93] Neurolog. Centblt., 1884.

[94] Char. Ann., 1885, p. 67.

[95] Jahrb. b. Kinderheilk., Bd. xxviii. p. 101.

[96] Ophthal. Rev., xv. 191.

[97] Berl. klin. Woch., 1884, 38.

[98] Ibid., 1889, 33.

[99] Ibid., 1884, 38.

[illegible] Oppenheim[100] sees an [illegible] migraine. As in the ophthalmic type [illegible] type, hemiparesis and aphasia [illegible] the lookout in such cases for hysteria.

[illegible] may complicate migraine, but the [illegible] been noted in ophthalmic [illegible][101] had a patient who [illegible] nerve, lasting three days, and [illegible] disappearance of the menses. [illegible] hysteria. Jacobi[103] has reported [illegible] of the right third nerve, which [illegible] and the pupil: the latter, while [illegible] to light. There was no migraine, [illegible] In studying hysterical affections [illegible] to confuse paralysis with [illegible] common.[104] Hysterical affections [illegible] are very numerous and not a little [illegible] may simulate the eye symptoms of migraine. This is true especially in cases in which there is an hysterical [illegible] blepharospasm or a spastic [illegible] of the muscles supplied by the third nerve, or possibly the sixth nerve.

Hysterical blindness [illegible] affection: in this condition there is [illegible] common affection is a contraction of the [illegible] a scotoma to a careless observer. In hysteria there is also a reversal of the normal color fields, the field for red being larger than the field for blue. These hysterical affections of the visual fields are always associated with other hysterical stigmata, and they are essentially unlike the visual aura of ophthalmic migraine. In hysteria there is not the scintillating scotoma, and the disorder in the visual field is rather a contraction than a true scotoma. In some very rare cases (one of which has been put on record by the present writer[105]) a true hemianopsia has, however, been observed in hysteria. But the visual aura in migraine, as we have seen, is very transient, differing in this respect from the visual disorders of hysteria.

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[100] Char. Ann. 1890, Bd. xiv.

[101] Hinde and Moyer, N. Y. Med. Rec., 1887, 418.

[102] Prag. med. Woch., 1883, No. 10.

[103] Am. Journ. Obstet., 1876, p. 223.

[104] Gilles de la Tourette, Hysterie, chap. ix. The subject is discussed at great length by Tourette, who seems to be sceptical about a hysterical paralysis of the *interior* muscles of the eye.

[105] Article on Hysteria in Dercum's Text-Book of Nervous Diseases, by American

As Tourette has pointed out in great detail, following especially Borel,[106] the muscles of the eye in hysteria are not so much the seat of paralysis as of spasm. In fact it may be a question whether there is a true hysterical paralysis of the third nerve, and whether the reported cases have not really been instances of blepharospasm and of spastic strabismus. In that curious affection known as monocular diplopia, in which the hysterical patient sees double with one eye, the cause is probably a spasm of the muscle of accommodation, causing irregular contraction of the crystalline lens.[107] So in the pseudo-paralysis of the third nerve, studied especially by French observers, the affection is in reality a contracture of the orbicularis muscle causing an apparent drooping of the upper lid. Blepharospasm, or contracture of the orbicularis, is indeed the commonest hysterical affection of the eye-muscles. Isolated paralysis of any of the orbital muscles, and especially of the intraocular muscles, as of the iris or ciliary muscle, of hysterical origin, is probably never seen, although apparent cases have been reported; but the strabismus is probably always due not to a paralysis but to a *spasm* of one or other of the ocular muscles. This seems to be the conclusion of Tourette, who has traversed this subject with great care, and to whose work the reader is referred for details.

From what has been written it is seen that the distinction between the ophthalmoplegia of migraine and the blepharospasm and spastic strabismus of hysteria is not a subject of great difficulty in the hands of a careful observer.

PSYCHICAL MIGRAINE.

Mental disorders of various kinds have been reported by many observers of migraine. The subject was discussed at length by Liveing,[108] who gave many instances which he had culled from the literature.

Authors, p. 112. In that article the hysterical affections of the eye are discussed in more detail than there is room for in the present paper. See Wecker et Landolt (Traité Comp. d'Ophthalmologie, article on Amblyopie Hysterique) for references to a number of cases of hysterical hemianopsia.

[106] Affections Hysteriques des Muscles Oculaires, Ann. d'Ocul., Novembre, Decembre, 1887.

[107] Tourette, op. cit. Also Parinaud, quoted by Tourette.

[108] Op. cit., p. 107. Liveing was a most diffuse writer and a great compiler, and some of his quotations are perhaps wide of the mark; but his passages on the psychical disorders of migraine form an interesting contribution to this subject, and more complete than any other work of which the present writer has knowledge.

He divided these disorders into two groups, the intellectual and the emotional.

Lebert in describing his own symptoms said that his seizures began with some incoherence of ideas and difficulty in finding words. Others have described confusion of intellect and impairment of memory. Even a partial loss of consciousness has been experienced. Some of these affections of mind are accompanied with a sensation of numbness in the extremities, face, and tongue, and with an affection of the speech, which will be referred to again. Some permanent impairment of mind has been observed in migrainous patients who have suffered with severe and oft-repeated attacks. Elaborate descriptions have been indulged in by some of these patients, as one old man, referred to by Liveing, who said that as the visual phenomena passed off, his memory usually failed so much that for a time he was mentally incapacitated, and whatever he read or did during that period left no impression: he experienced a singular disorder of ideation: circumstances and events of long before were brought back to him as if actually present: his consciousness appeared to be doubled, and the past and present were confused.

Emotional disorders are not uncommon during the first stage of migraine. The state of mind in some patients is one of fright or great dread. They know what is in store for them and fear it; but the apprehension or demoralization seems in some cases out of proportion to the cause. This has been acknowledged by migrainous patients themselves.[109] The visual aura, with its ugly scintillating scotoma, is particularly disturbing, and quite unnerves some individuals; so much so indeed that they avoid speaking or even thinking of it during the intervals, as in a case referred to by Airy. This vague and unaccountable fear supplies another analogy with epilepsy, in which, as is well known, a sense of terror is often experienced during the brief aura.

As we have said before, this mental state of the migrainous patient is well adapted to excite hysterical symptoms, and it is rather remarkable that more has not been written on this topic. Possibly some of the anæsthesias, tinglings, and speech defects are truly hysterical, but there is a dearth of cases in which the subject of hysterical stigmata has been put to the test. The severe suffering of the patient, following so soon on the aura, may mask such symptoms, and does not invite to a close examination for them.

Mental depression, not only during the aura, but even between the aroxysms, is present in some patients. It may be explained by the great suffering and the frequent incapacity which results from the attack.

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[109] An early writer who recognized this inordinate dread was Dwight, who gave a good description. (Med[illegible]sitory, vol. ii. p. 16, New York, 1800.)

In endeavoring to explain the mental symptoms in migraine two points of view present themselves. First, the psychical disorder may be due to the dread of the approaching suffering and to the suffering itself. Great pain, frequently repeated, is demoralizing to the human soul. The ancients said that physical pain was the greatest evil that man could endure. Second, the mental symptoms may be looked upon as an integral part of the nerve storm. This seems to be true from the fact that strong-minded men have endeavored in vain to control the emotional depression, the fear and anxiety, which sometimes precede the onset of the pain. In other words, there is a true psychosis attendant upon the attack of migraine.[110]

Disorders of speech have also been noted by not a few observers as occurring in migraine. Liveing[111] has collected much curious information on this subject. Oppenheim[112] says that he has observed aphasia, combined with agraphia. Word-deafness has been observed in only a few cases. The disturbance lasts from a few minutes to half an hour, and is succeeded by the headache, which is always upon the *left* half of the head. This fact, of course, suggests that the speech defects are due to a temporary affection of the speech centres in the left cerebrum. What this affection is must be as yet a mere matter of conjecture; but it seems necessary to suppose that it is some material affection, something acting to disturb possibly the blood supply or the nutrition of the brain cells of the speech centres in the cortex. Liveing, although he wrote before much was definitely known about the speech centres, saw the possible significance of these speech disorders, and collected a number of cases which tended to show that the migraine in these instances was on the *left* side of the brain. He noted that migraine is often a one-sided affection, and the thought occurred to him to investigate whether the left side was most often affected in these cases. Thus he found that in Lebert's own case the speech disorder was accompanied with a numbness in the tongue and fingers of the *right* hand. In several other cases the phenomena were similar; there was speech disorder, with numbness on the right side, as in the hand and arm, neck and face.

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[110] Oppenheim, Dis. Nerv. Syst., p. 751. The subject has been recognized also by Griesinger and Krafft-Ebing. The psychosis according to these observers is one of excitement, confusion, or stupor.

[111] Op. cit., p. 93. Localizing data are still wanting in migraine. See *ante*, p. 10.

[112] Op. cit., p. 751.

In Abercrombie's case the numbness (aura) extended upwards from the fingers of the *right* hand, and was followed by impairment of speech. Some of these cases were very striking, and seem to be more than mere coincidences. Of 15 cases thus analyzed by Liveing, in 7 the numbness was on the *right* side; in 4 it was bilateral, while in the remaining cases the report was defective. But in no single instance was the *left side of the body alone affected.* The inference seems clear that the speech defects in migraine are often caused by a left-sided lesion in the brain.

In some cases reported as migraine with aphasia it is possible that Bright's disease may have been a factor, for this affection may cause temporary aphasia with headache. The writer has seen a well-marked example, simulating tumor of the brain, in his service at the Philadelphia Hospital.

But if we are to follow out the analogy of migraine with epilepsy, we should seek for a true *psychical* migraine, not in the mere presence of psychical symptoms, but in the *substitution* of such symptoms for the classical migrainous attack. Are there such cases of pure psychical migraine? The answer seems from a few recorded cases to be in the affirmative. Sir George Airy [113] observed in his own person the usual attack complicated with transient impairment of speech and memory without either headache or numbness. Parry [114] advanced the theory of a *compensation* between the different phenomena of the seizure; if the disorder of sight proved abortive, the higher cerebral faculties were more likely to be involved. Tissot, quoted by Liveing, related a case in which attacks of habitual migraine were at length completely replaced by fits of disordered ideation—a true substitutional attack, or pure psychical migraine. We are indebted to Liveing for more information on this obscure subject than is supplied by most later writers.

Among other mental changes is the state known as double-consciousness.[115] Vertigo is also sometimes experienced. These symptoms indicate how profoundly the mental or cerebral functions may be affected in migraine.[116]

Treatment.—The treatment of migraine is entirely symptomatic and expectant. There is no specific for the disease. In our ignorance of the pathology of the affection, our treatment can be nothing more than empirical; and this fact is proved, as in all similar cases, by the large number of remedies that are recommended and even lauded in excess. From quack nostrums to spectacles the list is a dreary one, and will not be repeated here.

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[113] Philosophical Magazine, July, 1865.

[114] Pathology and Therapeutics, vol. i. p. 357, quoted by Liveing.

[115] Gowers, Dis. Nerv. Syst., vol. ii. p. 842.

[116] See also Bounal for a case with epileptoid symptoms, Rev. Mens. de Méd. et Chir., 1878, ii. 279; Gray, The Correlation and Interconvertibility of Migraine and Epilepsy, Pathologist, 1881, i. 4.

We may, however, consider briefly a few of the more approved remedies. From the writer's personal experience in his own case it has seemed that an abortive treatment is always worth a trial. This treatment consists in prompt action on the gastro-intestinal tract. A saline laxative, such as Rochelle or Epsom salts, taken at the first appearance of the visual aura, will sometimes cut short an attack. If free purgation is obtained in an hour or two, the attack may be aborted or its severity diminished. The same is true of an emetic, especially if the attack comes on soon after the patient has taken a hearty meal. Under such circumstances I have known a full dose of ipecac promptly to abort the attack. As soon as the stomach is emptied the hemicrania diminishes and soon ends. Although the remedy is not pleasant, it is pleasanter than a long period of sick headache with the inevitable emesis that comes anyhow at the termination of the attack. It is but proper to say, however, that in some of the more severe cases these remedies addressed to the stomach and bowels will not bring entire relief.

The symptomatic treatment is usually addressed to the relief of pain; and here the extensive list of anodyne drugs comes into play. A popular migraine powder at present is composed of citrate of caffeine, monobromide of camphor, and acetanilid. The various coal-tar products are often appealed to, but seldom do more than assuage the pain. They are not curative, and they are therefore often disappointing. Among them are antipyrine, antikamnia, and phenacetine.

Cannabis indica was long regarded with favor in migraine, and it is extolled by Sinkler[117] in his excellent discussion of treatment. Of recent years, however, we hear little of it. Remedies which are supposed to control the vasomotors, such as ergot, have also been commended, but on purely theoretical grounds. The victim of migraine who puts his trust in them will suffer for his faith. Nitrite of amyl is said to give relief in the early stage. The bromides have been used extensively, not without temporary relief.[118] So, too, of chloral, and croton chloral. Opiates, and especially the hypodermic use of morphia, will deaden the pain, but the practice of giving morphia in this way to patients who have frequent attacks, is most objectionable. The risk of forming the habit is not small. Local applications of all sorts have been used; heat, or its converse, cold, is probably among the best.

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[117] Article, Migraine, in Pepper's Syst. of Med., vol. v. p. 413. Also, by the same author, in Hare's Syst. of Pract. Therapeutics, vol. iii. p. 375.

[118] Tourette et Blocq (Prog. Méd., xv. 476), in a case of ophthalmic migraine with transitory aphasia, obtained good results with bromides, which they used in doses as high as 5 grammes a day.

The expectant treatment, or that which is applied to warding off the attacks, consists merely in a tonic, hygienic, and restorative regime. The usual, and sometimes useful, drugs for this purpose are quinine, iron, nux vomica, arsenic, and phosphorus; but it is the irony of therapeutics to recommend these drugs as in any sense curative.

Some patients, from long experience, find some particular drug or method best suited to their own cases, but the fact that such approved remedies are not universally effective proves how much the personal equation rules in migraine as in other neuroses. It is still a disease that favors experimental therapeutics, and the stage of scientific achievement has yet to be reached.

TETANUS.

The eye-symptoms in tetanus are few in number. In the ordinary form of the disease these symptoms are practically *nil*, but in that rare and interesting kind of lockjaw known as *cephalic* tetanus (the *kopf-tetanus* of the Germans) there is occasionally seen a paralysis of one or more ocular muscles.

In cephalic tetanus the disease usually takes its origin in a wound situated in the distribution of the fifth nerve. The tetanic symptoms may be limited to the head, throat and neck, although in some cases there is also opisthotonos, and even general tetanus. The peculiarity of this form of tetanus is that there is an associated paralysis of the seventh nerve. The muscles of deglutition are involved, a fact which led Rose to call the disease *tetanus hydrophobicus*. But this is an unfortunate name, for the affection has no relation whatever to hydrophobia.

Cephalic tetanus is a rare affection. But few cases have been recorded in America. The most complete account of the disease ever given in this country was published by Willard[119] in 1895. That report was based upon the history of a case in a boy, whose attack was caused by a wound, from a dirty stick, in the neighborhood of the orbit, and who made a good recovery.

Willard collected the references to 74 cases of cephalic tetanus, his own being the seventy-fifth case. There were 57 males and 15 females noted. In 45 early cases, in which the tetanus developed within the first week, 39 died and 4 recovered. In 32 later cases, in which the symptoms developed after the first week, 8 died and 24 recovered. These figures illustrate the fact that the mortality from cephalic tetanus is rather less than from the ordinary form, and that the prognosis is much more favorable in the late cases.

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[119] Tetanus Cephalic, etc., Trans. Col. Phys. of Philada., 1895, 3d Series, vol. xvii. p. 27.

The ocular muscles have been involved in a very few of these cases of cephalic tetanus. In the first place, of course, the orbicularis muscle, which is supplied by the seventh nerve and is not properly an ocular, or at least an orbital, muscle, is involved along with the facial muscles supplied by the facial nerve. The eye is open, as in all cases of peripheral palsy of the seventh nerve, a fact which shows that the paralysis is peripheral. There has been some discussion as to the exact pathology of this peripheral palsy; in some cases examined post-mortem the evidences of neuritis have not been found; and during life the reactions of degeneration have not been marked. In Willard's case the present writer studied the electrotonus, and found no reactions of degeneration, although the examination was incomplete owing to the patient's excitement and resistance.

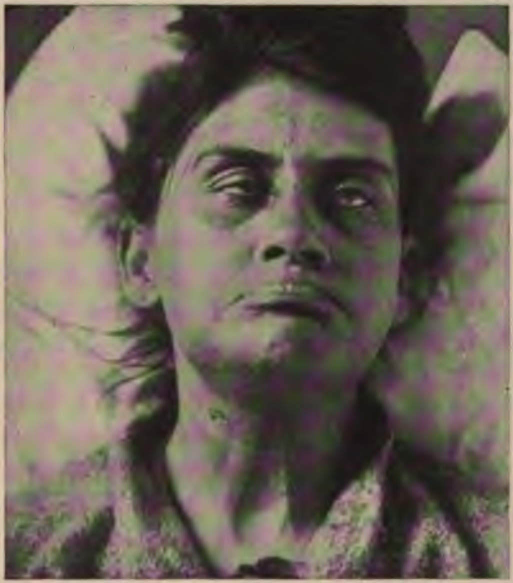

FIG. 3.—Cephalic tetanus, with paralysis of both seventh nerves. This picture shows the patient in the attempt to close her eyes. Trismus. Ironed-out expression. Wound on nose. The eye muscles were not involved, although the right eyeball rolled unduly inward in the attempt to close the eyes.

In a very few cases of cephalic tetanus some of the orbital muscles have been involved along with the facial muscles. In one case ptosis was observed.[120] In another case [121] paralysis of the external and internal ocular muscles of one side, with facial paralysis of the opposite side, was seen. Willard makes no reference to this complication, so it is probable that it has existed in rare instances only. V. Schrötter [122] has put on record a case of kopftetanus in which there was paralysis of the facial, the ocular, and the hypoglossal nerves. Neumann [123] says that the facial nerve is paralyzed first; then the oculomotor, abducens, trochlear and hypoglossal are affected in the order named.

The pathology of this affection, as already said, is obscure. Tetanus is caused by a bacillus, and this is no doubt so also in the cephalic form. But ordinary tetanus does not cause paralysis; quite the reverse. We are driven to the conclusion, entirely speculative, that for some reason the poison of tetanus, especially when introduced in the area of the fifth nerve, has the power of paralyzing some few of the cranial nerves, especially those supplying the face and the orbit, but not exclusively these, for the hypoglossus also may be involved. It is possible, as has been suggested to the minds of several observers, and has been voiced by Neumann, that the poison acts upon the nuclei of the nerves involved. This seems to be shown by the absence of the post-mortem evidences of neuritis, as well as the absence of the reactions of degeneration.

TETANY.

In this disease, which is not common in America, eye symptoms seem to be rare. Of recent European writers Filia,[124] who reports 44 cases, makes no mention of such symptoms. The same is true of Pons.[125] Another observer, Loos,[126] has observed nystagmus in a child. The fundus oculi is usually normal, although Müller [127] described a case with neuro-retinitis and later beginning atrophy; but the case was complicated with scarlet fever. V. Jaksch [128] had a case with double choked disk and diplopia, but other symptoms pointed to an organic brain lesion. Marschner[129] describes a case of neuroretinitis in tetany, but the patient had also pleuro-pneumonia. Cassel[130] describes a case of the disease with optic atrophy, but gives no other details regarding it.

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[120] Serein, L'Union Méd., 1886, No. 173.

[121] Roberts, Lancet, July 11, 1891.

[122] Fall von Kopftetanus, in Wien. klin. Woch., 1902, No. 2, p. 56.

[123] Ein Fall von Kopftetanus, in Zeit. f. Heilkunde, 1902, p. 344.

[124] La Tetanie chez les Enfants, in Rev. d'Hyg., 1903, ii. 505–537.

[125] Sur Quelques Points de la Tetanie Infantile, Toulouse, 1903.

[126] Deut. Arch. f. klin. Med., Bd. l. S. 169.

[127] Deut. Milit. Arzt. Zeits., 1884, xiii. S. 439.

[128] Zeit. f. klin. Med., 1890, xvii. 144.

Kursmal[131] observed a case in which the pupils were unequal. There is sometimes a cramp in the eye muscles. This is usually a blepharospasm. There may be also a sudden convergent strabismus with diplopia. The tetanic spasm of the eye muscles may be accompanied with pain, reddening of the conjunctivæ, and lachrymation.[132] Cases have been described in which the eye symptoms appeared alone, or alternating with the other symptoms. The pupil is seldom dilated, and even more rarely contracted.[133]

These few references to the disease, by recent European writers who see much of it (and much more than is seen in America) seem to indicate that the ocular symptoms, although not lacking, are rather rare. American and English writers (Osler, Dana, Gowers) make but little if any reference to these symptoms, and only to the contractions in the eye muscles.

EPILEPSY.

The ocular symptoms of epilepsy are important from a diagnostic stand-point. They have also in some cases not a little medicolegal significance. In considering them it is necessary to remember that they are usually of a transient character, and that to detect them requires alertness and some skill in observation. (See Chapter XVIII, p. 782.)

These symptoms may be divided conveniently into groups. Thus they are *visual*, *muscular*, and *vascular*. It is desirable to state at once that it is not the object of this paper to describe epilepsy itself, but merely its ocular symptoms. Only so much of the disease will be referred to as is necessary for an understanding of these symptoms. Epilepsy, like migraine, is an explosive neurosis, preceded in the vast majority of cases by an aura. Its ocular manifestations are sometimes a part or even the whole of the aura; while in other cases the involvement of the eye is in the nature of a sequel. In the majority of cases, however, it is either as an aura, or as a manifestation of the early stage of the fit, that the eye symptoms are prominent.

The purely *visual* symptoms of epilepsy are of course entirely subjective, and we must therefore rely entirely on the patient's own statements for a knowledge of them.[134] They may appear as an aura or as a sequel. It is in the nature of the auræ of epilepsy, unlike those of migraine, to be very fleeting, even instantaneous; and when these auræ are visual they consist usually of mere momentary affections of sight. Some of them are not unlike the visual auræ of migraine; a fact which has led some observers to see a more intimate relationship between these two diseases than perhaps the facts warrant.

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[129] Deut. Arch. f. klin. Med., 1896, Bd. lvi. 501.

[130] Berl. klin. Woch., 1896, 69.

[131] Deut. Arch. f. klin. Med., 1869, vi. 481.

[132] Hochwart, Tetanie, in Nothnagel's Spec. Path. u. Ther., Bd. xi. p. 134.

[133] Verein f. Psych. in Wein, 1895.

Mere obscuration of vision, something like the amblyopia of migraine, is an epileptic aura in some cases.[135] A true scintillating scotoma, somewhat like that seen in migraine, is claimed by Benswanger [136] to appear sometimes in epilepsy, but this is rare; and it is difficult to understand how an epileptic patient would have time ordinarily to note the characteristics of such a sign. Féré [137] also refers to obscuration of vision as a symptom of epilepsy. Some patients say that everything becomes black before them just before the seizure; by this they probably indicate a visual aura, or amblyopia. Herpin,[138] who has reviewed the history of epileptic auræ from the time of Hippocrates, gives no instance of a true visual aura, except in a case reported by Maissonneuve,[139] in which the attack began with a dazzling sensation (éblouissement) in the right eye, which continued for some minutes after the return of consciousness. The original idea of an epileptic aura was that it was caused by a "vapor" extending up the vessels of a limb; and indeed the word "aura" is derived from the Greek word meaning "a vapor." This idea was so firmly fixed that it dominated the medical world, as Herpin shows, even until very modern times, and to it may be ascribed the fact that the older observers were not on the lookout for any kind of aura except the sensory warning beginning at an extremity. Hence auræ of the special senses were not recognized by them, because, forsooth, they were not mentioned by Galen and the ancients. But in recent times auræ of the special senses have been fully recognized, and, as Gowers [140] remarks, a visual warning is twice as common as all the other special sense auræ together. This visual aura may be a sudden loss of sight, or it may be a flash of colored light or other chromatic appearance.

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[134] Spratling (Epilepsy and its Treatment, p. 226) gives a long list of the visual auræ which he has noted in his patients.

[135] Voisin, Les Epilepsies, 1896.

[136] Die Epilepsie, in Nothnagel's Spec. Path. u. Therap., Bd. xii.

[137] Les Epilepsies, Paris, 1890.

[138] L'Epilepsie. Paris, 1852, p. 389.

[139] Cited by Herpin, op. cit., p. 411.

[140] Dis. Nerv. Syst., vol. ii. p. 739.

Flashes of light or balls of colored or uncolored light are among the commonest visual auræ of epilepsy. They are variously described by patients, and doubtless they vary in different cases. There may be merely a glare, a flash, or a dazzling sensation of light, and this may be colorless or it may be distinctly chromatic. Red seems to be the commonest color. In some cases these brilliant auræ take distinct shapes, especially that of a ball, and this may be seen approaching or receding, growing larger or smaller. This tendency to increase or diminish in size, to advance or recede, is sometimes seen in other objects, as will be referred to presently. Gowers,[141] who seems to have had the fortune to observe more complicated cases of visual auræ than most other observers, describes instances in which these visual warnings were associated with other auræ, such as of taste, smell, and common sensation. He also calls attention to the fact that the various colors of the spectrum are not seen in the same frequency. Thus they appear in the following order, red, blue, green, yellow, and purple, which is not quite the physiological order in which the colors are represented in the normal visual fields, for blue occupies the larger field in the normal eye. There is some suggestion here of what occurs in hysteria—that is, a reversal of the color-fields—red occupying the most prominent place instead of blue. Voisin also says that red is the color most frequently seen in the epileptic aura.

A curious form of epileptic aura is that in which objects appear to grow larger or smaller, as the case may be. In the one case they may appear to approach, in the other to recede, or even to grow thicker or thinner. These phenomena have been called respectively *macropsia* and *micropsia*.[142] Patients have described the objects in a room as receding to a great distance. In other cases they appear to approach and enlarge until consciousness is lost in the fit.

Sometimes, as Féré points out,[143] there is a photophobia or *dysæsthesia* of the retina: the patient cannot bear the light, and there is a sense of pain in the eye with a forcible reaction of some of the ocular muscles, especially a blepharospasm. Féré and others also refer to a dyschromatopsia, or inability to distinguish colors; but it is difficult to understand how such fine tests can be made in the brief period of an epileptic aura. Dyschromatopsia and changes in the visual field, such as contraction and reversal of the color-fields, may persist as post-epileptic phenomena, and these must carefully be distinguished from hysteria. In fact, various hysterical stigmata may complicate cases of genuine epilepsy, as the present writer has noted in the Philadelphia Hospital more than once.

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[141] Epilepsy and Other Chronic Convulsive Diseases, 2d Ed., p. 74 et infra.

[142] Veraguth, Ueber Mikropsia und Makropsia, Deut. Zeit. f. Nervenheilk., Bd. xxiv., H. 5, 6.

[143] Op. cit., p. 78.

The visual aura is sometimes located by the patient on one side, as in a case reported by Féré,[144] in which the patient saw a red mist before the right eye. In such a case it is a question whether the phenomenon is not hemiopic,—that is, seen in the corresponding halves of both retinæ. For instance, when the object is seen apparently only by the right eye, it is really perceived by the *left* half of the retina in each eye.[145]

Spratling[146] speaks of a *retinal* epilepsy, in which there is sudden blindness of brief duration without convulsion; in other words, a sort of substitutional attack. The term "epileptic amaurosis," used by Hughlings-Jackson, has also been used by some writers in this sense.

Of all visual auræ the most complex are the true hallucinations, in which, as it were, a panorama is unfolded before the patient's gaze. A few such cases have been reported. Gowers[147] describes minutely a complicated aura in which the patient saw an old woman in a brown dress, and Gregory's patient saw an old woman, too, but she was dressed in red.[148] These visions are sometimes of disagreeable or ugly objects or animals; but occasionally the scene recalls events of long ago, as is sometimes seen in migraine also. There may be hemianopic hallucinations.[149]

The *muscular* affections of the eye in epilepsy may be observed in either the extra- or intraocular muscles or in both. It is not unusual in the epileptic fit for the eyes to be turned forcibly in one direction, as, for instance, upward or to one side. When the eyes are strongly rotated to one side it is usually in cases in which there is forced rotation of the head also to one side, and the eyes are usually rolled towards the same side as the head. Onuf[150] claims that there are exceptions to this rule, and that the eyes may be rolled to the opposite side from that towards which the head is rotated, but such exceptions must be rare. This lateral deviation of the head and eyes in epilepsy may possibly indicate that one cerebral hemisphere is more involved in the epileptic discharge than the other, and it is the opposite of what is seen in gross cerebral hemorrhage in which there is also lateral deviation of the head and eyes, but *towards* the lesion,—*i.e.*, *away* from the paralyzed side. In epilepsy the deviation is usually *towards* the more convulsed side.[151] This is shown in cases of post-hemiplegic seizures in which the paralyzed side is the seat of the most active convulsions. Lateral deviation of the eyes, however, is not a localizing symptom of any great value in cerebral pathology. It occurs in too many conditions to be of special significance.

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[144] Op. cit., p. 464.

[145] Harris (Hemianopsia, in Brain, 20, 307) refers to temporary hemianopia in epilepsy.

[146] Epilepsy and its Treatment.

[147] Epilepsy, etc., 2d Ed., p .78.

[148] Cited by Benswanger, op. cit.

[149] Harris, op. cit. See also *ante*, p. 11.

[150] New York State Med. Journ., 1904, iv. 71-74.

Diplopia has been reported by some writers as occurring in the early stage of epilepsy. Féré and Gowers both speak of it. Strabismus and nystagmus have also been noted, both as accompaniments of the attacks and as sequellæ of them. Diplopia is doubtless caused by a temporary strabismus which results from either a spasm or paresis of one or other ocular muscle. When it occurs as a part of the epileptic aura it is probably due to a spasm of some of the ocular muscles causing the two eyeballs to roll in different axes. Féré[152] relates a case in which an epileptic boy became suddenly cross-eyed in the interval between fits; this convergent strabismus, in the left eye, was increased after each seizure, at which time the patient saw double. The same author has noted permanent lateral nystagmus, also permanent blepharospasm following epileptic seizures. Such permanent affections of the ocular muscles point to organic lesions, whether in the nature of an accident of the fit or as a cause of the fit, such as a tumor or a basilar meningitis. Speaking of nystagmus, Féré says it occurs *towards* the affected hemisphere in hemiplegic epilepsy in the initial stage.

Much has been written about the state of the pupils during the epileptic fit.[153] It is commonly said that the pupil is dilated and unresponsive to light. Onuf says that there is extreme contraction of the pupil just as the attack begins, and that it is also contracted in the post-convulsive stage. Gowers confirms this statement with respect to the initial contraction, but says that dilatation quickly supervenes and is present during the tonic stage. He found, however, that in mild attacks the pupil is not always unresponsive to light. Reynolds[154] points out that at the end of the fit the pupils often exhibit marked oscillations. These oscillations consist of alternate contraction and dilatation, but are not present in every case. The conjunctival reflex is also abolished during the profound unconsciousness of epilepsy: the eye may be touched or irritated with the finger without causing the usual reflex contraction of the orbicularis. In very mild cases of petit mal this may not always be so, doubtless because consciousness is not lost, or only momentarily so.[155]

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[151] Féré, op. cit., p. 387.

[152] Op. cit., 129, also p. 177 et infra.

[153] Féré, op. cit., pp. 89, 178, 391, 417, 473.

[154] Epilepsy, 1861, p. 112.

The condition of the pupil has been appealed to in some cases as a medicolegal test. Evidently, in simulated epilepsy this state of dilatation could not be feigned by an act of the will; but, as Voisin has pointed out, dilatation can be secured by the use of belladonna, and deception might thus be practised. Still, the action of belladonna or its alkaloid atropia is very persistent on the pupil, lasting for days, and this should be enough to expose the deception.[156] The dilatation in epilepsy, with abolition of the light-reflex, does not endure after the fit. But in a suspected case the dilatation with immobility of the pupil *during* the fit, speaks for epilepsy.[157] Féré speaks of asymmetry of the pupil in the epileptic fit; also of inequality of the pupils.[158] It is important to bear in mind that we are here discussing essential or idiopathic epilepsy,—*i.e.*, epilepsy without gross organic lesion. In cases of symptomatic epilepsy, as, for instance, in cases of tumors, hemorrhage, or meningitis, the state of the pupils might be affected by such lesion more or less permanently and independently of the seizures.

Vascular lesions in the eye are not common in epilepsy. Ecchymoses or extravasations of blood beneath the conjunctiva are occasionally seen as results of the violence of the fit.[159] I have seen such a case in which a large part of the exposed surface of the eyeball was covered with extravasated blood. It is not a symptom of special importance, and it usually disappears in a few days. Congestion of the conjunctiva may be present during the seizure, but it soon subsides. These ocular hemorrhages are sometimes associated with hemorrhages in other parts of the body.[160] Retinal hemorrhage is rare. Gowers thinks it is prevented by the intra-ocular tension. The appearance of the fundus is not significant in ordinary essential epilepsy. Between attacks the fundus is normal, and during attacks the only marked change is increased fulness of the retinal veins; this is in unison with the general congestion of the vessels of the head. When traces of old optic neuritis are found in so-called idiopathic epilepsy, they may be due to an old syphilitic lesion which may have acted as the original cause of the epilepsy, as Gowers has pointed out. Epilepsy itself does not cause permanent changes in the fundus of the eye, as is the universal verdict. It is a rather curious fact that, as Féré points out, an ophthalmoscopic examination in an epileptic will sometimes excite a seizure.

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[155] Spratling (Epilepsy and its Treatment, 1904, p. 268) does not recall a single instance in which the pupils were not more or less dilated—in some cases extremely so. He does not believe that this is due to asphyxia, as some have held, because he has seen it in light attacks as a very early phenomenon. He thinks that inequality of the pupils points to an organic lesion, and thus it may persist during the intervals.

[156] See the Trial of Allen C. Laros, which took place at Easton, Pa., in August, 1876 (reported by Edgar), for an example of alleged feigned epilepsy in which the state of the pupils was discussed.

[157] Max Bire, Ueber Epilepsie, Deut. Zeit. f. Nervenheilk., Bd. xxiii., H. 1, 2, p. 36. See also Hoche, Die differential Diagnose Zuischen Epilepsie und Hysterie, Berl. Augen. Huschweld, 1902.

[158] Op. cit., 391.

[159] Voisin, op. cit.

In convulsive attacks due to uræmia and to chronic lead-poisoning (encephalopathy) there may be well-known changes in the fundus due to those conditions, but such changes are in nowise either the causes or results of the fits.

There are but few ocular *sequellæ* of epilepsy. Mention has already been made of ocular palsies which may persist after the fit, but they are rare. In genuine psychical epilepsy, in which the convulsion is replaced by a psychosis, hallucinations of sight may occur; also in the post-paroxysmal stage.[161] These hallucinations are usually of alarming objects, as ugly faces, devils, etc.[162] In testing the visual fields after an epileptic attack, it is important to remember, as already said, that such symptoms as dyscromatopsia, contraction of the visual fields, reversal of the color fields, etc., may be of hysterical character, for it is not rare for epilepsy to be complicated with hysteria. The error must not be made, however, of supposing that because a patient has a few hysterical stigmata after a fit, therefore his attack is not genuine epilepsy. The two diseases may co-exist in one and the same patient. So, too, of migraine. The fact that a patient has visual auræ resembling those of migraine, or even the still more striking fact that a patient has both migrainous and epileptic seizures, is no reason for claiming that those two diseases are so intimately related as is done by some authors, conspicuous among whom are both Gowers and Féré.[163]

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[160] Spiller, Uncinate Group of Fits, Am. Med., 1904, vii. 474.

[161] Sachmunde, Ueber vereinzelt auftretende Halluzinationen bei Epileptiken, Monat. f. Psych. u. Nerv., 1904, xv. 434.

[162] Voisin, op. cit.

[163] H. C. Wood (Medical News, 1894, p. 707) has reported a case of epilepsy with migrainous symptoms. Such a form might appropriately be called *migrainoid* epilepsy. Hughlings-Jackson (Lancet, 1875, vol. ii. p. 244) spoke of migraine as

THE BLINDNESS DUE TO URÆMIA AND OTHER BLOOD-STATES.

Under this heading will be described those attacks of blindness, usually sudden and sometimes transient, which are observed occasionally in uræmic patients, also in anæmic and other blood-states, and likewise those due to embolus of the retinal artery. This chapter, however, does not include in its scope an account of the permanent changes in the fundus oculi due to Bright's disease or to other organic diseases in the optic nerves and brain.[164]

Uræmic Blindness.—Sudden blindness sometimes occurs in uræmic patients. It is usually seen in cases of kidney disease that are well advanced, although cases have been reported in which the amaurosis was an early symptom. But, whether early or late, the amaurosis is usually a sign that the renal disease is severe, and the inference is that the system is profoundly poisoned. The characteristics of this blindness are its sudden onset, its completeness, the preservation of the reactions of the pupil, the sudden disappearance of the blindness when the blood-state is relieved by active purgation or sweating, and the absence of ophthalmoscopic changes, excepting such as may previously have existed.[165]

The other symptoms associated with this uræmic blindness may or may not be marked. In many cases there are no prominent brain symptoms of uræmia, no coma, no convulsions, or any special signs of uræmic encephalopathy. In other cases the visual phenomena precede a more active and extensive attack of uræmic-poisoning. There may be some headache and mental confusion, which pass away with the blindness under active treatment, or the case may advance to convulsions and coma. Most of the cases put on record have been those in which the blindness was the prominent or only symptom. It is probable that some affection of vision precedes most attacks of uræmic convulsions and coma, but is overlooked in the gravity of the succeeding symptoms.

There has been much speculation about the causes of the sudden and transient blindness of uræmia. Most observers are agreed that there are no ophthalmoscopic changes to account for it, or that such

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"sensory epilepsy," and he had a rather fanciful theory that it is due to a "discharging lesion of the occipital lobe." There is no scientific warrant for such a pathology or such a terminology.

[164] Antonalli, L'Amblyopie Transitoire, in Arch. de Neurolog., 1892, pp. 202, 423.

[165] Gowers, Manual of Medical Ophthalmoscopy, 1904.

changes, if present, are slight. Gowers says that slight, transient œdema of the papilla has been noted to coincide with the symptom and to pass away with it. This is also noted by Orters.[166] In some cases, however, even this œdema of the nerve-head has not been seen.[167] Considering the frequency of retinal changes in chronic Bright's disease, it is rather remarkable that no alterations are reported in so many cases of sudden blindness. Gowers does, indeed, say that permanent retinal changes are present in some cases, but that these, while, of course, they persist after the attack, are not changed by it. It might well be difficult to detect transient changes under such circumstances.

It is natural to fall back upon the theory of an acute poisoning of the nerve fibres, without visible organic changes. This is a reasonable theory, in accord with the condition of the patient, and in accord also with what we know about acute poisoning of nervous tissue. A toxic substance in the blood might well affect the nutrition and function of the optic nerve and retina without giving signs that could always be recognized with the ophthalmoscope. Thus the blindness might be due to the overwhelming of the nerve with the uræmic poison.[168] This is the *chemical* theory,[169] according to which deleterious substances are circulating in the blood, though what the exact chemical composition of them is remains unknown.

Another theory is the *mechanical* one, according to which the blindness is due to an œdema of the brain and of the optic nerve and retina.[170] We have already referred to the fact that œdema of the nerve-head has been noted by various observers. It has been argued by Hauenschild that œdema of the nerve-head alone could not cause the symptoms, for the reason that the pupillary reflexes are preserved; therefore, the lesion must be located back of the primary optic ganglia, possibly in the brain cortex. This is an ingenious argument, and not easily answered. Certainly, if the pupils react to light, the retinal cells must be capable of taking impressions and sending them on to the ganglia; in other words, the functions of the optic nerves are not entirely gone, as the total blindness would seem to indicate.

This mechanical theory, or theory of pressure due to œdema, is much the same as the *vasomotor* theory, which here, as elsewhere, has played its part. This is, however, a most unsatisfactory theory by which to account for anything in pathology, for although the vasomotors are undoubtedly a means by which disease processes act, they nevertheless must require some cause to set them in action. It is this cause, whatever it may be, acting upon the vasomotors, that is the true cause of any disease process, not the vasomotors themselves. A poison can conceivably affect the vasomotors, as can also mechanical irritation or a gross lesion, but nothing is explained by merely appealing to the state of the vasomotors so far as essential causation is concerned. Pal,[171] discussing the pathogenesis of acute transitory amaurosis of uræmia, eclampsia, etc., says it is due to a transitory anæmia of the brain due to vasomotor change, cardiac insufficiency, etc. This seems like begging the question. Certainly, cardiac insufficiency is not present in most of the cases of puerperal eclampsia, in which, just as in uræmia, some of these affections of sight are observed. Benschoten suggests that the blindness may be due to a spasm of the retinal vessels. Bull[172] suggests that arteriosclerosis of the ophthalmic and internal carotid arteries may be the underlying cause of some cases of obscure blindness, especially shown by a paracentral scotoma.

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[166] Dobrowolsky, Klin. Monatsbl. f. Augenheilk., March, 1881, p. 121.

[167] Hauenschild, Muench. med. Woch., 1903, No. 4.

[168] Benschoten, Providence Med. Journ., July, 1903.

[169] Frerichs, Arch. f. Physik. Heilk., x. 34.

[170] Traube, Schmidt's Jahrbuch., cxiv, p. 308.

In uræmic amaurosis there is usually an obscuring of the whole field, and equally in both eyes. But Pick[173] reports a case in which there was an homonymous hemianopsia of uræmic origin.[174]

Uræmic blindness has been observed in various forms of kidney disease. In Hauenschild's case there was a parenchymatous nephritis. The condition may occur in contracted kidney also. Roscher[175] reported a case in which there was a suddenly developed hemorrhagic nephritis; two days later uræmic symptoms appeared, with amaurosis. In this case there were normal eye grounds, but the pupils were dilated and would not react, contrary to what is usually reported. This case was rapidly fatal, and Roscher believed that the immobility of the pupils suggested a bad prognosis, because it indicated the severity of the process.

In puerperal eclampsia there are eye symptoms exactly like those seen in uræmia. Disturbance of vision is observed in many cases; there may be amblyopia or amaurosis. The late Professor Parvin[176] referred to a fatal case of eclampsia occurring at the fourth month of pregnancy in which there was almost total blindness for two days before the seizure. Dewees [177] many years ago said that a temporary loss of vision was among the premonitory symptoms of puerperal convulsions. Of recent years this symptom has been so generally recognized as not to require us to quote many authorities. Neither is this the place to enter upon a discussion of the question whether eclampsia is identical with uræmic convulsions.[178] Certainly it resembles the uræmic fit in many of its symptoms, among which is amaurosis.

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[171] Centblt. f. inn. Med., 1903, No. 17.

[172] Ann. Ophthal., 1904, xiii. 74.

[173] Ueber Hemianopie bei Uraemie, Munch. med. Woch., 1903, p. 2125. This condition was first described by Pick in 1896. A few cases have been described by others.

[174] See also Gill, Transient Recurrent Attacks of Lateral Hemianopsia, in Brit. Med. Journ., Feb. 1, 1890, p. 233.

[175] Muench. med Woch., 1903, No. 9, p. 382.

[176] Eclampsia, in Hirst's Am. Syst. of Obstetrics, vol. ii. p. 73.

Anæmia from hemorrhage and other causes may produce sudden blindness. This blindness may be trifling or complete, transient or permanent, immediate or postponed, and more frequent from so-called spontaneous than from traumatic hemorrhage. The pupils are usually dilated and immobile to light. It has been observed after hemorrhage from the bowels, uterus, nose, as well as after venesection and trauma. The visual disturbance may be in the nature of a scotoma, or hemianopsia, or contraction of the fields, or the blindness may be in sectors or irregular.[179] There may be changes in the fundus, or this may be normal. Benschoten says that the prognosis is good if the blindness is immediate, but unfavorable if it is delayed several days, because in the latter case it may be due to neuritis or atrophy of the optic nerve. He believes that the prognosis is worst in cases of hemorrhage from the stomach. Assicot [180] reports two cases of permanent blindness due to hemorrhage from the uterus. In transitory blindness due to anæmia Loewe [181] supposed that there was a spasm of the blood-vessels supplying the cuneus, a theory to be taken for what it is worth. Transient loss of sight occurs also in syncopal attacks.

In diabetes there are sometimes observed defects of sight which are similar to uræmic amaurosis.[182] Lecorché called attention to the grave significance of diabetic amblyopia in some cases, in which it may be a terminal symptom, or at least herald the end.[183] It should not be forgotten that cataract occurs in some cases of diabetes.

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[177] Midwifery, 3d Ed., Philada., 1828, p. 451.

[178] Winckel, Obstetrics, Am. Trans., p. 587 et infra.

[179] Gowers, op. cit.

[180] Pathogenie des Amauroses Posthemorrhagiques, in Arch. de Ophth., May, 1902.

[181] Ein Fall von Transitorischer Blei Amaurose, in Arch. f. Augenheilk., xlviii. p. 332.

[182] Gowers, op. cit. See also Heine, Deutsch. med. Woch., 1903, p. 348; and Brunton, in Reynolds's Syst. of Med., vol. iii. p. 591.

[183] Traité du Diabète, 1876. Also Laveran et Teissier, Pathologie Médicale, 3d ed. vol. i. p. 314.

Jacquean has even written of amblyopia of hepatic origin.[184] Amblyopia from tobacco and alcohol is usually due to a post-orbital optic neuritis, and is not to be described in this chapter.[185]

Finally, brief reference may be made here to embolus of the retinal artery as a cause of sudden blindness. This blindness may be permanent or it may tend to improve. The causes are such as give rise to embolism in other arteries, especially diseases of the endocardium; and there may be also arterial sclerosis with temporary stoppage of the circulation. Emboli and thrombi may occur in these cases in other arteries in the brain.[186] In a case observed by the present writer in an old lady the blindness was sudden, complete, and permanent, and the embolus was doubtless derived from the heart, which was diseased. The ophthalmoscopic changes are usually well marked, and consist in a diminution in the size of the artery and, in time, atrophy of the disk and retina.[187]

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[184] Bull. et Mém. Soc. Franc. d'Ophthal., 1902.

[185] De Schweinitz and Edsall, Concerning a Possible Etiological Factor in Tobacco and Alcohol Amblyopia, etc., in Am. Journ. Med. Sci., 1903, p. 216.

[186] Posey (Transient Monocular Blindness, in Journ. Am. Med. Asso., May 31, 1902) has described cases in which the blindness was evidently due to vascular defects other than embolic, as the loss of sight was only temporary. In some cases there was actual sclerosis.

[187] Norris, Medical Ophthalmology, in Pepper's Syst. of Med., vol. iv. pp. 740, 741. See also, Van Duyse, Arch. d'Ophth., 1902, p. 93.

CHAPTER XVIII.

NEUROSES AND OTHER CONDITIONS OCCASIONED BY ERRORS OF REFRACTION AND IMBALANCE OF THE EXTRA-OCULAR MUSCLES, AND THE EFFECT OF LENSES AND OPERATIVE PROCEDURE IN RELIEVING THE SAME.

PART I.

BY S. D. RISLEY, M.D.

HEADACHE; NEURALGIA.

THE design in the present chapter is to treat primarily of headache and its relation to eye-strain; but it will not be found possible to avoid a more or less extended consideration of a group of associated phenomena composing the symptom complex known as "asthenopia."

In the discussion of the subject it will be maintained—

(1) That in a large number of patients anomalies of the ocular apparatus are the sole and sufficient cause of headache.

(2) That these anomalies of vision may be the unsuspected cause and, therefore, that the absence of symptoms obviously referable to the eyes does not exclude them as an etiologic factor in headache and certain associated symptoms,—*e.g.*, insomnia, vertigo, petit chorea in children, stomach derangements, and impaired general nutrition.

(3) That the congenital origin of these ocular anomalies does not exclude them from participation as a factor in the sudden, or recent, development of symptoms coming on during, or after, middle life, after attacks of acute disease, or more or less acute exacerbations of some general dyscrasia.

(4) That, therefore, the participation of the eyes as a probable etiologic factor in headache and other neuroses can be positively excluded only in the absence of ocular disease, or after the most painstaking correction of any existing error of refraction or abnormality of binocular balance.

(5) That in many cases the prolonged eye-strain, consequent upon existing anomalies of vision, sets up pathologic conditions of the *fundus oculi,* and that these require time for recovery, therefore, immediate relief of symptoms is not always to be expected.

The facts set forth briefly in the preceding propositions may be claimed as among the most signal achievements of modern ophthalmology. While it is true that a number of early observers had recognized a more or less definite association between weak eyes and pain in the brow, they failed to grasp the true relationship of the symptoms. Indeed, any historical study of the early literature of headache, or even of much that has been written by later authors, is disappointing when viewed from the standpoint of the ophthalmologist. In the general or specific discussion of this symptom and its etiology by some authors, the eyes as a probable factor are noticed in such a casual manner that it is obvious no adequate comprehension of the importance of eye-strain had been reached. To the ophthalmic surgeon this practical neglect of ocular affections as a cause of headache and a considerable group of associated nervous symptoms is unaccountable, since in the daily routine of his work he encounters it in so large a percentage of his eye-strain patients. In 1899, the writer made a cursory analysis of one thousand consecutive eye patients, and found that upward of 50 per cent. of them complained of headache, and that many of them had been sent by their physicians in the hope that the pain in the head, which had proved rebellious to general treatment, might be due to some ocular defect. A colleague, Dr. M. W. Zimmerman, in an elaborate study of two thousand patients, drawn from private and hospital cases, found that 71.3 per cent. of them suffered from headache. In ophthalmic practice, therefore, the subject assumes a place of commanding importance.

Until within the memory of men now living, and actively engaged in professional work, ophthalmic literature was no less at fault. We are, nevertheless, indebted to many of the early writers for painstaking and accurate descriptions of the symptom complex we now know as *asthenopia*. The weak eyes, which baffled all their efforts for relief, were associated by them with brow pain, and were carefully described by Taylor in 1776; by Plenck in 1792; by Scarpa in 1816, and by Beer in 1817 (Donders). These observers were followed, prior to 1850, by Lawrence, Tyrrel, Sichel, MacKenzie, and others. The variety of opinions expressed by these eminent men and the many designations devised and employed as either descriptive of the observed conditions or explanatory of their supposed nature or cause convict them of error. This is not surprising since it was not possible to form any adequate conception of the essential nature of the morbid conditions they observed until after the discovery of the hypermetropic eye, and the publication, in 1858, of the justly renowned physiologic researches of Donders of Utrecht in his classic treatise on the "Anomalies of Refraction and Accommodation." This remarkable book shed a flood of light upon many ocular problems, and laid the foundation for a most important part of the beautiful structure which modern ophthalmology has reared.

This great master does not, however, seem to have comprehended the full clinical significance of the truths he had discovered and established. The ulterior results of the strain upon the accommodative function of the eye in overcoming the anomalies of refraction, and of the struggle required to maintain binocular single vision in the presence of some abnormality of the extraocular muscular apparatus, have for the most part been disclosed by his followers. The credit for recognizing the great significance of eye-strain in producing both the pathologic states of the *fundus oculi*, and an associated group of general reflex symptoms, often simulating organic disease, is largely due to American observers. Dr. William Thomson, in 1879,[1] in a paper on "Astigmatism as a Cause for Persistent Headache and Other Nervous Symptoms," states that more than ten years before he became aware that persons who consulted him for defective sight presented symptoms which he enumerated as pain in the brow, temples, and occiput, a sense of fulness in the head amounting to vertigo and nausea, insomnia, loss of appetite, fear of impending apoplexy or epilepsy, general nervous prostration, choreic twitching of the muscles of the head and face, all of which he had seen relieved by correcting their astigmatic eyes. Other ophthalmic surgeons had also witnessed the marked relief of headache, insomnia, and other reflex symptoms, following the correction of optical defects by glasses.

Dr. S. Weir Mitchell's attention was called to these cases, and to him is due the credit of impressing upon the general profession, in two forcible articles,[2] the grave significance of eye-strain. Ever since attention was thus fixed upon the subject, in Philadelphia at least, no study of a rebellious case of headache or of obscure nervous disease has ever been considered complete until a careful examination of the eyes had excluded them as a possible cause of the disturbance.

It does not fall within the scope of this chapter to consider in detail the nature and variety of the ocular anomalies which may cause an undue strain or tension upon the visual organs, but it is important to have a definite conception of the normal or essential requirements for comfortable binocular vision. That is to say, a physiologic standard to which anomalous states may be referred for comparison.

This standard will be found in two healthy emmetropic eyes with

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[1] The Medical News and Library.

[2] Medical and Surgical Reporter, 1874; American Journal of the Medical Sciences, 1876.

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[illegible] the eyes is sup-
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[illegible] be indicated by

the frequent distortions of the human skull, as shown by any cursory study of a hatter's forms, many of which are so ill shaped as to appear grotesque when compared with the diameters of a model skull.* One need not go far to verify the fact that these deformities of the skull cause, in many persons, variations in the size and form of the bony orbits, and this leads to the more or less serious deformity of the developing eyeballs, which the orbits are designed to inclose and protect.

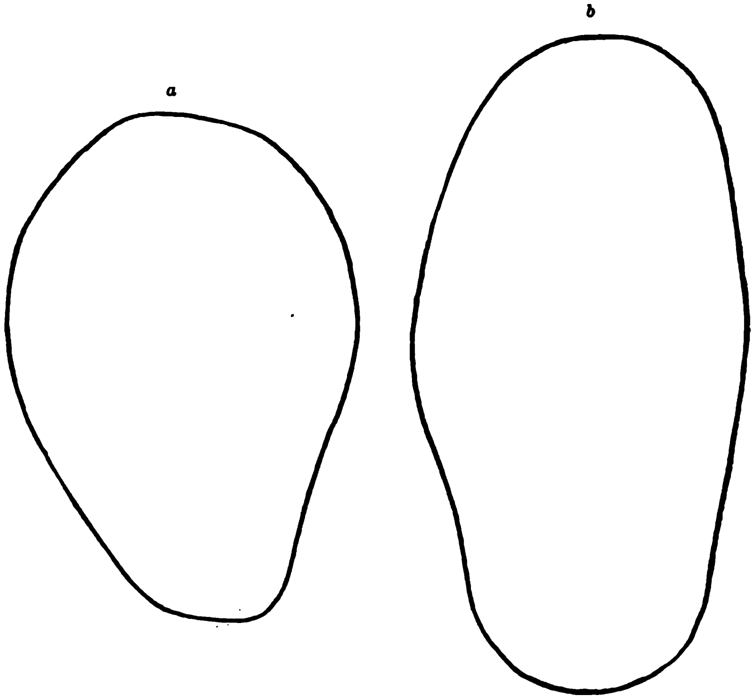

FIG. 1.—Diagrams showing types of conformation of skulls in subjects whose eyes were highly astigmatic, with asymmetrical meridians, associated with binocular imbalance.

It is not unreasonable to expect that, during development in an anomalously shaped orbit, not only certain modifications will occur in the shape of the eyeball itself, which would change its diameters and alter the radii of curvature of the cornea, but would lead also to changes in the length and line of direction of the optic nerves, and the origin and attachments, the length and direction of the ocular muscles,—conditions one or all of which would lead to more or less marked disturbance of the refraction and motility of the eye, and in many complex ways modify the conditions of binocular vision rendering it either difficult or impossible. (Compare Chapter XIV and XXIII.)

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*"Abnormalities of Ocular Balance," by S. D. Risley, M.D., University Med. Mag., Jan. 1895.

[illegible]

[illegible] For example, the [illegible]

[illegible] a great variety [illegible] meridians.

[illegible] of the Eye, edited by Norris [illegible]

—*i.e.*, the meridian of least defect is vertical in one eye and inclined in the other or irregularly placed in a great variety of ways. 4. One eye is astigmatic, the other emmetropic or has a simple hypermetropia, or both are hypermetropic, but in different degree (anisometropia), or both are myopic with or without astigmatism. 5. One eye is hypermetropic with or without astigmatism, the other is myopic with or without astigmatism (antemetropia). 6. Both eyes are myopic with or without astigmatism, which, as in hypermetropic refraction, may be symmetrical or not, or present in different degrees between the two eyes. 7. In both hypermetropic or myopic eyes with astigmatism the meridian of least defect may be "against the rule,"—*i.e.*, horizontal instead of vertical, symmetrically or asymmetrically inclined from the horizontal.

When compared to the emmetropic or model eye all of these complex conditions are to be regarded as defects. They are essentially anatomical anomalies. When viewed from the standpoint of physiologic optics they constitute the errors of refraction in the eye.

The emmetropic eye has an average length of about 22 millimetres and its dioptric system will focus parallel rays of light on the retina, at the fovea, without any accommodative effort; that is to say, its far-point is infinity, its principal focal point lies on the retina. Such an eye has a range of accurate vision from infinity to the physiologic near-point. The near-point gradually recedes from the eye until at forty-two to forty-five years of age presbyopia is reached. Any departure from this ideal state of refraction must sooner or later introduce disturbance of function. In the routine of practice it simplifies the theoretical considerations to reduce the anomalous states of refraction, as enumerated above, to the ideal standard (emmetropia) by means of carefully selected glasses, which, when placed before the eyes, neutralize the defective conditions. To accomplish this would seem at first sight a simple problem in mathematical optics, but the human eye has been provided with the power of accommodation through the agency of which it is able to change the focus of its dioptric system. This power makes it possible even in the presence of anomalies of refraction, to so change its dioptric system as to focus more or less accurately the image of surrounding objects upon the retina. This power resides in the ciliary muscle and is exercised involuntarily. It follows that in any effort to measure the static refraction of the eye, this ever changing power of accommodation becomes an unknown quantity and must of necessity vitiate the results obtained. (Compare Chapter II.)

It is not difficult to detect the presence of the grosser forms of these congenital defects, but they do not, as a rule, comprise the most important group in their relation to headache and reflex symptoms. Where binocular vision is impossible because of the gross nature of the muscular anomaly, or where the existing error of refraction is too great or of such a character as not to be overcome by any muscular effort, the afflicted individual perforce resigns to the inevitable, gives up the struggle, and is usually free from pain. It is quite different, however, when the existing error is of such a nature that the efforts of the individual can overcome it in the interests of clear vision. It is this effort which constitutes the eye-strain and is the cause of intra-ocular congestion, weak eyes, and pain,—in a word, of *asthenopia*.

In the case of grosser defects of vision diagnosis is not difficult, since they are accompanied by impaired vision or by a manifest tendency to double vision. Obscurity exists only in the case of defects of minor degree, or when the experience and habit of a lifetime has taught the individual the trick of rendering the congenital ocular defect latent; that is to say, of covering it up by a tonic muscular tension. In such cases the vision with both eyes or for each may be normal, or approximately so; the range of accommodation and binocular balance may be but little if at all deranged, the existing congenital abnormality in this group of cases revealing itself only to careful analysis by the skilled observer. It becomes necessary in all such patients to temporarily suspend the action of the ciliary muscle before any trustworthy determination can be made of the static refraction of the eye.

This is accomplished through the instillation of the so-called mydriatics, or cyclophlegics. Notwithstanding the now well-known fact of the very high percentage of defective eyes and the very generally recognized relation between these defects and headache, the discussion of the subject should be approached with great conservatism.

That eye-strain and certain diseases of the eye—*e.g.*, chorioiditis—are in many persons the sole and sufficient cause for headache can no longer be questioned in the light of experience, since the ophthalmologist is a daily witness of the complete relief from the malady afforded by the correction of errors of refraction and abnormalities in the ocular muscles or by the cure of intra-ocular disease. Experience, however, has shown that, in other groups of patients suffering from headache, there may be no relation between existing ocular defects and the pain in the head, since the most careful correction of the existing anomalies affords no relief. Some examples of migraine belong to this class. All headaches are not due to eye-strain. It is not a very unusual experience to remove the eye-strain by glasses to the great relief of ocular symptoms, without any appreciable result in relieving the headache;

or, certain kinds of pain in the head, in any given patient, may be relieved and others still persist.

It must be borne in mind also that many patients suffer greatly from impaired vision, chronically inflamed and painful eyes, who never have had a headache or other reflex symptoms. It may be stated as an ophthalmic aphorism that when eye-strain manifests itself by red lids, recurring styes and symptomatic conjunctivitis that such persons comparatively rarely suffer from headache, while on the other hand the patients who suffer from reflex symptoms as the result of ocular defects may exhibit no local external inflammatory or irritative manifestations. There are numerous exceptions, however, to this statement. Then, too, the subject grows in complexity, from the standpoint of diagnosis, because of the fact that in two patients suffering from similar or identical ocular defects, one may have no symptoms other than weak eyes and a blurring page after prolonged effort, while the other is constantly in the slough of semi-invalidism from pain in the head and other reflex symptoms which, from exposure to light or the persistent use of the eyes, or from the general fatigue following some unusual exertion, culminate in more or less frequent and violent explosions of so-called "sick or nervous headache," and both of whom find complete and lasting relief from glasses so nearly alike that they might be used interchangeably. In a third case, with a similar ocular defect, there are no symptoms either reflex or local, the strain being borne with impunity. While then it is unquestionably true that in many instances the existing ocular defects are the sole and sufficient cause for their headache, the great difference in the effects of eye-strain, upon different individuals, in combating the untoward conditions presented by these congenital anomalies make it necessary to look beyond the eye for some adequate explanation. Sufficient cause for the phenomena may often be found in the difference of vocation, environment or temperament. For example, the persistent use of the eyes in one case may be unnecessary and in the other compulsory. One may be in good general health, the other a victim of hereditary or other neuroses, or some complicating diathesis which impairs the nutrition and introduces a tendency to pain and local pathologic states. Ambition to excel, or the sanguine temperament, may drive one into constant activity in spite of the pain occasioned by the use of the eyes, while the other rejects the goad of ambition unwilling to secure success or to court fame at the sacrifice of pain. Then, too, in the presence of ocular conditions causing eye-strain, other things being equal, the neurotic individual is more likely to suffer from headache and associated phenomena than those who cannot be designated as nervous people. While, therefore, the general conditions of health or temperament or the necessity for constant use of the eyes may in some sense be regarded as the cause of the headache, the primary factor in its etiology is the eye-strain, and the pain will not be prevented until the exciting cause is removed, although it may be partially relieved or modified by improvement in general conditions.

An extremely interesting group of patients are those who suffer from sick or nervous headache and general nervous symptoms,—*e.g.*, nausea, insomnia, irritability of temper, facial chorea in children, etc., and are completely and permanently relieved of their symptom complex by the correction of some ocular defect which throughout life has been the unsuspected cause of their suffering.

Many patients who suffer severely from headache demonstrably due to ocular defects or disease, are often quite free from local symptoms. If ocular symptoms are present during acute exacerbations of headache, they occur *pari passu*, or come on after the reflexes, and are regarded by the patient as due to headache. Patients often claim that their "eyes are weak because they have a bad head" and submit to examination of the eyes reluctantly and only on the advice of their physician, deeming it unnecessary since there are no obvious eye symptoms and therefore apparently nothing wrong with their eyes. In these patients the location of pain is often suggestive of the ocular origin of the symptoms. Vertex pain is comparatively rarely due to eye-strain. In optic-nerve disease, as in post-ocular neuritis and atrophy, there is in many cases pain in the front part of the head on top, but it is not a vertex pain. The pain when associated with the class of eye affections under discussion occurs in the brow, occiput or nape of the neck, back of the eyes and shooting thence to the occiput, as a hemicrania, or in the temples. In point of frequency the pain occurs probably in the order named. Simple eye-strain due to some error of refraction,—*e.g.*, hypermetropic astigmatism, particularly in children,—will usually give rise to brow pain. If the refraction error is associated with an imbalance of the ocular muscles so that constant muscular strain is required to maintain single vision, as in exophoria, a tendency for the eyes to diverge; or in esophoria, a tendency to converge; or in hyperphoria, a tendency of one eye to turn upward, there will usually be occipital pain also. If at the same time marked turgidity or inflammation of the chorioid, retinal irritation or disease of the retina and chorioid in the region of the fovea have been set up as the immediate result of the constant struggle for accurate vision, in the majority of patients there will be a tense post-ocular pain which shoots to the occiput, nape of the neck, or even down the spine, not infrequently radiating to the shoulders and arms. In two notable instances which caused great anxiety, one a busy attorney, the other an eminent physician, the writer witnessed complete relief from pain in the region of the heart, which shot backward to the left scapula, follow the correction of an astigmatism against the rule in the one, and correction of hyperphoria by tenotomy in the other.

Many of these patients, although unwilling to ascribe their suffering primarily to their eyes, nevertheless recognize the harmful influence of exposure to strong light, or, that exacerbations of pain come on after extraordinary use of the eyes, but others never do and ascribe their eye symptoms to "biliousness," or to overfatigue, hunger, and many other things.

Others regard their recurring sick headaches as hereditary and relate that one or both parents and other members of the family are victims to the same malady. They think, therefore, that they are born to a heritage of pain from which it is useless to seek permanent relief. I have seen many of these lifelong sufferers from recurring sick headache entirely and permanently relieved by the correction of their faulty ocular conditions. It is doubtless true that the neurotic tendency may be a family trait, but it is equally true, even more probable, that the anatomic defects already noticed, upon which the errors of refraction or the ocular imbalance depend, are hereditary. The regularity with which defective eyes,—*e.g.*, hypermetropia with astigmatism,—are handed down from parent to child is one of the most striking facts in ophthalmology. It should be noted in this connection how frequently it is to the form of the skull that the characteristic family likeness is largely due. It is highly probable that the defective form of the eyeball which produces the ametropia, and the faulty attachment of the muscles to the globe which causes the ocular imbalance, are the direct results of the defects in the shape of the bony orbit, and this in turn is due to distortion of the skull. It follows, therefore, that the possibility of hereditary anatomical malformations should be excluded in every case before accepting the theory of hereditary headache. (See Page 954.)

There is much variety of manifestation in different patients even in the presence of similar ocular defects. Many suffer almost constantly from brow pain and pain in the occiput. It is quite common for patients afflicted with muscular anomalies to have their rest disturbed at night by a dull, dragging pain in the back of the head and neck. Many patients complain that they are often awakened in the night by this distressing symptom and get relief only by shifting the position of the head. One patient remarked that he always awoke in the morning feeling as though he had had a billet of wood for a pillow. He had in addition to his astigmatic eyes a hyperphoria and was not relieved of his nocturnal headache and insomnia until vertical balance between the eyes was restored by tenotomy, notwithstanding the fact that his weak eyes and headache had been relieved during his waking hours by glasses containing a prism which corrected the hyperphoria.

The complaints of a busy physician who consulted the writer in the hope that his eyes might afford some clue to the origin of his symptoms furnish a vivid illustration of the unsuspected participation of the eyes. He suffered from almost constant occipital pain, considered himself a chronic dyspeptic, was in despair over the insomnia which for many years had been his constant terror. He had consulted many colleagues in different cities without lasting relief, and finally was advised to abandon his work for a year and take a long sea voyage on a slow sailing merchantman. It was on his return from this trip, which had consumed more than six months, that he consulted me. He had left home "a nervous wreck," had returned much improved in many ways, but no sooner had he entered upon his work as a rhinologist than he suffered a speedy return of all his symptoms. He enjoyed normal acuity of vision in each eye, and had no subjective eye symptoms. The ophthalmoscope revealed marked retinal irritation, however, the fundus being uniformly red and fluffy. There was a low grade of heteronymous astigmatism, a tendency for the two eyes to converge, and a right hyperphoria of 3°. The refraction error was carefully corrected under prolonged mydriasis, and subsequently the hyperphoria relieved by tenotomy. Very little relief followed the use of the correcting glasses, but as soon as the vertical deviation between the eyes was relieved by operation his symptoms promptly vanished, not only the headache and insomnia, but the dyspepsia. He ate with impunity, the morning following the operation, "a hearty breakfast of sausage, buckwheat cakes, and coffee," and continued for years to eat, without subsequent harm, anything he wished. The general health improved and he has continued his work with satisfaction.

Many persons, although rarely free from dull pain in the head, suffer more or less acute exacerbations after exposure to light, from prolonged use of the eyes at near-work, after an evening spent in the theatre, or by general fatigue, but their symptoms are never violent. Others, while like these, are rarely free from pain, are subject to steadily progressive aggravation of all the symptoms until they culminate in violent attacks which drive them to bed in a darkened room, from which they emerge after twelve or twenty-four hours with a dull sore head, and weak eyes, which are tender to palpation and unduly sensitive to light.

The explanation for the advancing symptoms up to the point of crisis is found, not only in the muscular fatigue and exhaustion of the nerve-centres, but in great measure is due to the steadily increasing hyperæmia and turgescence of the uveal tract of the eyes coming on under the strain of near-work or by exposure to light. It is an interesting fact that the acute symptoms often present themselves many hours after the culminating cause. For example, an evening at the opera will be followed by the acute attack the following morning after a restless or sleepless night, the insomnia being due to the ocular turgescence. The relief comes after the subsidence of the ocular congestion, which results from the confinement in a darkened room, the emesis, the administration of a saline cathartic, and the cold compresses over eye and brow, which are the forms of treatment ordinarily adopted by the patient, and which experience has taught are the most efficient means of relief. They return to their usual vocation only to pass through the inevitable cycle of suffering. Many patients expect these attacks of headache once a week at least, and return from a journey or shopping expedition in the full expectation of spending the next twenty-four hours in bed.

A striking illustration of this group of patients was afforded by a young lady, a guest in the writer's house. She arrived worn out by a long journey. The following morning she was unable to appear at breakfast, and was found in the throes of a violent sick headache. The eyes were red, the lids swollen, she had photopsies which she described as wheels of fire and circling balls of red light. The pain was more severe in the right side of the head. She related that she had suffered these attacks two or three times every month since adolescence; that when the attack was imminent she usually awoke in the morning with dim vision, scintillating flashes of light and zigzag "wall of troy" lights in the periphery of the field of vision, which at times would present all the colors of the rainbow. These would repeatedly narrow down to near the centre of the field, then widen out into broader circles, grow fainter and finally disappear, leaving her with obscured central vision, objects being seen as through a veil. This obscuration would last but a brief time, but was always followed by nausea and vomiting, and the onset of violent headache, usually a right hemicrania at first, which later spread to the entire head. Every noise or ray of light added to her suffering, the slamming of a door being especially excruciating, as it seemed to her to shake the entire house. The attacks usually forced her to remain in bed for the entire day, and her "head felt tired and tender" for a day and often two days after the subsidence of the acute symptoms. Her mother and an older brother had suffered in the same manner since her earliest recollection.

A solution of atropia sulphate was instilled immediately into both eyes and repeated at brief intervals. All other means of relief were omitted. In an hour the pain subsided, and in two hours she was able to come down stairs to my consulting room. I there found a hypermetropic astigmatism, not in high degree, but with the axis of the required correcting cylinder against the rule in the left eye, and diagonal in the right. There were absorption changes in the fundus of both eyes, and macular retinochorioiditis in the right, a fact which, together with the diagonal corneal meridians, explained the right hemicrania. After a few days of treatment she received a pair of correcting glasses. These she has worn for twenty years with no recurrence of her attacks of "migraine;" she has suffered rarely from a dull headache, but then only after special stress of circumstances or when her "glasses were fitting badly."

Another interesting example of this group of cases was furnished by a lady aged 49 years, who wrote from a distant city for an appointment. She desired a consultation at her hotel, as she wrote that she would be suffering too severely from "sick headache" after her journey to be able to leave her bed for a day or possibly two days thereafter. When the writer called at the appointed time he found her, as she had predicted, in the throes of a violent headache; nervous, exhausted, suffering from nausea and fruitless attempts at emesis. A mydriatic was immediately instilled, and cold compresses applied to the closed lids, and potassium bromide administered. The patient was completely relieved in an hour, rose, ate a hearty meal, and the following morning was able to go to the office of the writer. Here it was ascertained that she had a high grade of hypermetropic astigmatism. After this had been corrected by glasses she was permitted to return to her home. Upon her arrival she wrote that for the first time in her experience she had been able to enjoy a railroad journey and had reached her home in perfect comfort. Ocular defects are often the cause of headache after a journey by rail, in a carriage, or after shopping or mingling with crowds of people.

Closely allied to this group of patients are those who claim to be nervous and suffer from what they call nervous headache. They are rarely nauseated, but have violent pains with muscular twitchings and hysterical crises. They usually seek for relief in opiates and analgesics.

Such patients are generally women, but the writer recalls the case of a merchant, the father of a numerous family of children, and the head of a large mercantile house, who had high hypermetropic astigmatism and right hyperphoria. The right side of the face was smaller than the left, the right zygoma being flat, while the left formed an arch. The hypermetropia was higher in the right eye than in the left, and the principal meridians of the cornea were inclined 45° from the horizontal plane. The refraction defect had been partially corrected by glasses, but the ocular imbalance neglected. Suddenly, while at his place of business, he would have some obscuration of vision accompanied by shimmering, rainbow-colored phosphenes in the periphery of his field of vision. Taught by many experiences he would hastily summon a cab to take him to his home, for he recognized the phosphenes as an invariable prodrome of an attack of severe headache. His wife described to me one of these exacerbations.

Arrived at home, his fortitude entirely exhausted, he would ring the doorbell violently, enter the house holding his head between his hands, and often unable to reach his bedroom, would throw himself upon the hall or parlor floor in despair, and entirely beside himself in an agony of suffering, burst into a flood of tears, roar with pain, making the most violent manifestations, alarming his wife, children, and servants, until the doctor came and relieved him by a hypodermic injection of morphia. These violent attacks were relieved by careful correction of his refraction error and a subsequent tenotomy of the right superior rectus muscle; but he still suffers from a dull occipital pain after a "hard day." Since these attacks often came on after some fancied indulgence at the table, or after an evening spent at the theatre followed by a supper, he ascribed them to deranged stomach and liver, and was in consequence a victim to the cathartic habit.

These violent onsets of pain in the head are occasionally associated with more or less violent storms of the nervous system, hysterical crises, cataleptoid seizures, and in one case which came under my eye the attacks were followed by what had at least been diagnosticated by an eminent authority as epilepsy.

A man thirty-five years of age had for several years been under treatment for epilepsy, but suffered almost constantly from headache, which culminated in paroxysms of pain preceded by phosphenes, which had come to be regarded as the aura of his epileptic seizures. He was sent to me for blurring page, and in the hope that his headache might be relieved by glasses. I found a moderate degree of refraction error, corrected by $+1.50^{s}+.50^{c}$ ax. 180° in each eye, and a marked absolute insufficiency of the internal recti. He received correcting glasses for constant wear and a second pair combined with prisms, base in, for all continuous near-work. I did not hear from him again for eight years, when he returned from his distant home for some change in his glasses, and much to my surprise said that he had not had a single return of his headache or convulsions after the treatment of his eyes. He then described to me his former attacks. Hurrying to his home with the first appearance of the phosphenes, he would soon be in the throes of a violent headache. The paroxysms would culminate in loss of consciousness, general convulsions, frothing at the mouth and wounding of the tongue, after which he would fall into a profound sleep from which he could not be awakened, often lasting for several hours.

Another striking instance of headache associated with nervous symptoms was a tall, thin, pallid, nervous lad, with red hair, age 17. He was the son of a busy attorney who was overworked and in feeble health, and a nervous, hysterical mother who had mixed astigmatism. The father and a brother had high myopic astigmatism and chorioiditis. The boy was brought because of his weak eyes and severe headaches, often attended with nausea and vertigo. The day before his visit to me, the father had found him in an unconscious condition in an armchair in the library before breakfast, with his legs and arms extended and rigid, his head thrown back, the eyes wide open, and the morning newspaper fallen from his hands. While examining the eyes I had an opportunity to witness one of these cataleptic seizures, which came on while attempting to determine the range of accommodation. He had a high degree of mixed astigmatism and insufficiency of the interni. There was marked chorioiditis and the eyeballs were stretching; that is to say, the refraction was passing from hypermetropia into myopia through the turnstile of astigmatism. His eyes were subjected to prolonged treatment and finally corrected by glasses resulting in relief so nearly complete that he finished his college course and became a successful civil engineer. There can be but little doubt as to the relation of cause and effect between his ocular conditions and the storm in his nervous system. Although he received alteratives internally, and his general regimen was carefully regulated, all this had been done before by a wise and experienced family physician who still conducted the general treatment.

The group of patients illustrated by the foregoing examples, who had all been lifelong sufferers from headache and other neuroses, aggravated by any occupation involving the use of the eyes, are those had in mind by Gould in his Biographic Clinics, and render highly probable the truth of his contention, at least in the examples selected by him from famous literary folk, by way of illustration of his thesis. They all may without difficulty, so far as the symptomatology is concerned, be reproduced again and again from the case books of every ophthalmologist.

A class of patients, some of whom present great difficulty in diagnosis, are those who develop headache during and after middle life. The pain is periorbital, post-ocular, or occipital, and may present any of the characteristic features already described in the preceding groups. The pain may be dull and constant, more or less aggravated by using the eyes or by exposure to light, or they may have severe periodical exacerbations of pain. Any existing refraction error or other ocular defects have heretofore given no evidence of their presence, or the eye may be approximately emmetropic and the vision normal, and the patient nevertheless associate his pain with the use of his eyes. A careful ophthalmoscopic study reveals a maroon-colored fundus with pigment clouds, and fine granular changes in the region of the fovea and between it and the optic nerve. The nerve itself, while possibly clearly outlined, is too vascular and appears therefore too deeply colored. The history may be entirely negative regarding any recognized ocular disturbance in the past. There is a widespread feeling in the community that glasses should be avoided as long as possible, many persons taking pride in the fact that they are forty-five or even fifty years of age and have never worn glasses. The fact is that, given an emmetropic or model eye, the physiologic range of accommodation is so far diminished in most persons soon after forty years of age, that a weak convex glass for reading will afford both additional comfort and safety. When an extra innervation strain must be placed upon the accommodation to secure satisfactory vision in reading, certain anomalies of convergence are produced which disturb the ocular balance and all of the chain of phenomena depending upon eye-strain follow,—*e.g.*, headache, intra-ocular hyperæmia, and chronic irritation or inflammatory changes in the fundus. As representing this group may be cited the case of an eminent clergyman aged fifty. This gentleman had resting upon him not only the responsibilities of a large and influential parish, but often occupied the lecture platform, and was a frequent contributor to religious and literary periodicals and the author of several books. He began to suffer from insomnia and almost immediately became the victim of severe occipital pains and occasional attacks of vertigo, for which he sought the advice of his physician. A diagnosis of cerebral hyperæmia from overwork was made and a grave prognosis given. He was advised to abandon his work for a year and seek relief in foreign travel. Quite disheartened, he consulted the writer and told him of his plans. His symptoms suggested the possibility of eye-strain, and brought out the fact that he had never worn glasses notwithstanding his age, and the enormous amount of literary and other work he was doing. He received a pair of working glasses. His insomnia and headache disappeared promptly, and although arrangements had been made for a long vacation they were cancelled and he went on with his work. He was to all appearances in perfect health and was a noteworthy example of stalwart manhood. There was, therefore, no suspicion of any neurotic or other predisposing element entering as an etiologic factor, his symptoms unquestionably being the result of his neglect to neutralize by glasses the physiologic loss in his range of accommodation.

Another simple explanation for the sudden onset of pain after 40 years of age is the existence of minor errors of refraction which have given no recognized trouble until the diminishing range of accommodation has arrived at a point where the accommodation strain sustains such a relation to its total value that it can no longer be borne with impunity and hence the onset of pain and its attendant phenomena. The only source of relief for such patients is the use of carefully selected glasses. These two groups of patients fortunately comprise the larger percentage of patients between forty and fifty years of age who seek the advice of the ophthalmologist for the somewhat sudden onset of headache.

It must be borne in mind, however, that the loss of accommodation is due to a general diminution in the elasticity of the crystalline lens, a change probably indicative of like degeneration processes going on in the other tissues of the body and which culminate in old age. They have at least reached the age of beginning degeneration.

In many persons certainly, the wear and tear of life begins to manifest itself between forty and fifty years of age in tissue changes, often pathologic in character. The commencing affections of the general vascular tree are visible not infrequently to the magnified image of the details of the eye-ground furnished by the ophthalmoscope before any suspicion of their presence is afforded by any notable general symptoms; but the appearances in the fundus, while they awaken the suspicion of the ophthalmic surgeon, and may lead to a study of general conditions, are not always characteristic of any specific dyscrasia and may readily be ascribed to the irritation and other local changes caused by prolonged eye-strain, as described in the preceding paragraphs. It is therefore possible to err in diagnosis at this point, since the vascular optic disk, the general fundus conditions, and the headache may be a part of the symptom complex of some serious dyscrasia which is sapping the vital functions. The existence, therefore, of some refraction defect, or neglect of the timely use of suitable glasses for near work should not be too readily accepted as the sole cause of headache in patients who have reached middle age.

The following case will illustrate the need for great care.

Mr. X., aged 49 years, an artist who spent many hours daily in his studio, had for several years suffered more or less constant occipito-frontal pain with frequent paroxysms of nervous headache which forced him to bed. In 1902, following one of these attacks, he noticed a central blurred spot before the left eye. This proved to be due to a minute hemorrhage at the fovea. The fundus of both eyes was cinnabar red, and there were numerous dark pigment clouds in the periphery. The veins were too full and dark, the optic nerves dark red but not swollen, there were faint gray lines along the larger retinal arteries, and fine granular changes in the macular region of both eyes. He was using for his work weak convex spherical glasses, whereas he had in both eyes hypermetropic astigmatism requiring the axis of the correcting cylinders against the rule. Here was sufficient cause for his headache, and it is not uncommon to find similar fundus conditions due solely to the continued eye-strain to which his eyes had been subjected. The macular hemorrhage, however, and the gray outline of the arteries, which suggested endarteritis, could not be overlooked. He ascribed his trouble to an attempt to reproduce upon the canvas the glint from a bright sabre which was placed in such a position as to reflect a strong light. The effort had developed a severe paroxysm of headache. He considered himself in good general health. He had never eaten meat, having been reared as a vegetarian. There was no discoverable arterial atheroma, but he had an accentuated systole and any vigorous exertion caused dyspnœa of a mild type. His habits were sedentary. He often passed too great quantities of urine, and a specimen examined was negative but had a low specific gravity and a few hyaline casts. His health failed rapidly, an intermittent albuminuria developed, and he died in uræmic convulsions in a year. The acute symptoms came on while he was on a sketching tour in the mountains, and was ushered in by one of his paroxysmal headaches. The glasses he received had for some months modified his head symptoms, and doubtless the eye-strain was a factor in their etiology, but behind the local conditions lurked, unsuspected by himself or his family physician, the general dyscrasia which led to a speedily fatal termination.

Dr. K., aged 56, a dentist, became the victim of insomnia, dyspeptic symptoms and occipital headache with frequent attacks of vertigo. A diagnosis of "concealed gout" had been made, and his headache ascribed to cerebral hyperæmia. The general regimen had been carefully and wisely regulated, comprising a prescribed diet, horseback exercise, bathing, etc., together with lithia waters and drugs. In spite of an intelligent and rigid obedience in carrying out the prescribed rules laid down for his guidance, the symptoms persisted and drove him from his work daily with severe occipital pain. He was then advised to abandon his work and spend six months in Switzerland. He experienced marked relief and returned to his chair, only to suffer a speedy relapse. He consulted me then of his own volition about his eyes. He was unquestionably the subject of lithæmia, but that this was not the sole cause of his headache was demonstrated by a steady but rapid subsidence of his symptoms after the correction of an astigmatism which had been neglected in the glasses he had been wearing. He is now nearing seventy years of age and has steadily pursued the arduous duties of his profession without a return of his symptoms.

It is not unusual for a patient during or after attacks of acute disease,—*e.g.*, coryza, influenza, pneumonia, etc.—to become the victim of headache which persists and is aggravated into acute paroxysms by the use of the eyes. Examination reveals the fact that they are subjects of some congenital anomaly of refraction, which has for the first time manifested itself in the symptoms of eye-strain, under the lowered physical tone produced by disease, and they are never again able to avoid the use of correcting glasses.

There are numerous patients who never suffer severe pain as the result of their eye-strain, but are nevertheless victims of a group of symptoms which must be classed as ocular neuroses. Insomnia has been mentioned as a frequent symptom. The reverse of this is a common complaint. Many persons become drowsy, especially in the evening, and find it quite impossible to read or do any eye work without falling asleep. I have many times seen this relieved by a pair of glasses. A constant sense of general fatigue or malaise is often caused by eye-strain. A busy physician came for relief for some illy defined ocular disturbance. He was 49 years of age and used +2D. for reading. He received for constant wear the following formula for glasses:

$$\text{O.D.} + .62 \subset + 1.^{c}\ \text{ax. } 30^\circ\ \text{V.} = \frac{6}{\text{VI.}}$$

$$\text{O.S.} + 1.00 \subset + 1.^{c}\ \text{ax. } 150^\circ\ \text{V.} = \frac{6}{\text{VI.}}$$

with a reading segment added of + 1.75D.

He returned in a week to express his satisfaction and remarked that he had been chronically tired for a long time; had had his heart examined and an analysis made of his urine, fearing the incipiency of some organic disease, but that all these symptoms had promptly disappeared under the relief from his eye-strain.

The facial twitchings of school children, with brow pain, irritability of temper, uncontrollable restlessness, disturbed sleep, precarious appetite, and poor progress at school, all of which make them the despair of their mothers and teachers, have many times been relieved by a pair of glasses. Whether these cases can be properly classed as petit chorea or not, it is nevertheless certain that the eye-strain is for them "the thorn in the flesh" which produces a constant irritation of the nervous system. In another group, although the defective eyes are evidently an important factor in the causation of symptoms, they are obviously not the sole cause. The above symptom complex may be present, but there is in addition many evidences of a vicious metabolism. The eye ground is fluffy and turgid, with here and there areas of pigment absorption; the veins are large and dark, the lymph channels are engorged, the optic disk is too red and its margins obscured; the retrotarsal folds of the conjunctiva are thickened and opaque, the caruncles are swollen and red, they have blepharitis and frequently recurring styes. A careful and painstaking study will in many cases reveal an irregular astigmatism as shown by the ophthalmometer or by skioscopy. They suffer severely from frontal headache and undue sensitiveness to light; the anterior perforating blood-vessels are enlarged. No glass will afford a normal acuity of vision. The pain in the head, the restless sleep, the precarious appetite, the muddy skin, the many evidences of deranged alimentation and impaired metabolism, make it difficult to draw the line sharply as to the relative responsibility of local and general conditions. The eyes are unquestionably suffering from the strain at school in overcoming a congenital anomaly. The anterior segment of the eyeballs is stretching under the increased pressure from within, due to the hyperæmia and turgescence of the intra-ocular tunics, and this increased tension is doubtless in some measure the origin of the brow pain and other reflex phenomena; but it is doubtless true also that these local ocular conditions might not, and probably would not, have occurred, in many cases certainly, but for the predisposing influence of the impairment of the general nutrition. While, therefore, it would be a fallacy to ascribe all of this group of conditions to eye-strain alone, nevertheless the correcting glasses and prolonged rest from near-work often prove of great relief to both eyes and head. The sleep improves, the nervous symptoms disappear, and in many instances the improvement in general health is so speedy, following the treatment of the eyes, that the inquiry is suggested whether the impaired metabolism was not in large measure due to the restless sleep, and constant headache due to ocular conditions. Analogous states have been observed as due to pharyngeal adenoids and to phymosis, so that it is not unreasonable to suppose that similar general conditions may be produced by the pain and irritation of prolonged eye-strain, the consequent fatigue of the nerve-centres involved, and the turgid states of the intra-ocular tunics.

The headache and insomnia of aged people is not infrequently produced by similar conditions of the chorioid and retina, and rapidly disappear under suitable local treatment addressed to the ocular conditions alone. I have seen many examples of this, some of which excite commiseration. The following history is illustrative: Miss McA., aged 68 years, came for advice regarding her failing vision, inflamed eyelids and a troublesome nictitation. Vision was much reduced in both eyes, but was improved by convex glasses. She had hypermetropic astigmatism in both but much higher in the left eye than in the right. There was no complaint of pain. The caruncles were red and swollen, there was long-standing blepharitis, incipient cataract, floating vitreous web, and chorioiditis with macular retinitis were present in both eyes. Inquiry about her general health educed the reply that all her life she had been a victim of sick headaches. She recalled that as a small child she returned from school with red eyes and headache. Burying her head in her mother's lap she would burst into tears from the violent pain. These attacks had continued until fifty years of age, but since then, although she suffered more or less constantly from "neuralgia," the pain did not cause nausea or emesis as in former years. The neuralgia had been "better of late because of medicine from her physician," which proved to be Kenyon's neuralgia pills. So utterly was this lifetime of pain divorced in her mind from ocular participation that she did not think it necessary to mention it until the history was drawn out by inquiry. Yet the ophthalmoscope revealed the scars produced by years of eye-strain, which was finally culminating in impaired nutrition of the vitreous body and the crystalline lens. The neuralgia pill was omitted, homatropine instilled four times daily, and smoked glasses with complete rest of the eyes advised. In a few days the external redness and nictitation disappeared, she slept soundly, the appetite improved, and a sense of discomfort and tension about the head vanished.

It is therefore important in both children and adults who are obviously the subjects of impaired general nutrition and suffer from headache that the ill health should not be too rigorously regarded as the sole cause of all their symptoms.

In a large percentage of persons whose headaches are without question due solely to eye-strain and its associated local conditions, the symptoms disappear promptly and permanently under correcting glasses, but in many others the result is disappointing. It has already been pointed out that as a result of protracted eye-strain pathologic states of the fundus oculi are produced. The turgid, fluffy chorioid, absorption chorioidal crescents at the temporal margin of the optic nerves, granular changes at the fovea with impaired acuity of central vision are conditions witnessed daily in the consulting room as the outcome of constant strain required to overcome the existing congenital defect. The congestion of the vascular chorioid produces increased intra-ocular tension and pressure upon the terminals of the ciliary nerves, resulting in headache and the other reflex symptoms under discussion. The tender, readily-yielding sclerotic coat of childhood often yields to this increased pressure from within; the eyeballs stretch and pass from hypermetropic refraction into myopia. It is to these conditions that the increasing percentage of near-sight among school children is due, and statistics have shown that in this group is found the sufferers from headache, restlessness in school, blepharitis, weak eyes, and impaired acuity of vision. The distention of the globe is not always uniform, sometimes falling upon the posterior pole as in many cases of high myopia, but often affecting the anterior segment, especially in the ciliary region, which becomes thin, appears bluish in color, and is traversed by the dilated anterior ciliary blood-vessels which perforate the globe in this locality to anastomose with the long posterior ciliary arteries in the ciliary region of the uveal tract. It is in these cases of change in the curvature of the anterior portion of the ball that variations in the corneal astigmatism are observed. Now it is not to be expected that these conditions, which gravely involve the nutrition of the entire globe of the eye, will vanish at once under the use of correcting glasses. More or less protracted rest from use at a near-point, avoidance of exposure to strong light, careful attention to the general regimen, and, more than all, the passage of sufficient time for recovery, are all required to effect relief from symptoms and a cure of the pathologic states primarily set up by the eye-strain.

Taken at the onset of symptoms the results might have been prevented. These are the conditions which lie at the foundation of the modern requirement that no child shall be permitted to enter upon school life until examination of the eyes has either excluded the existence of these congenital defects or secured their correction by glasses.

The same chain of results may occur in adults without the existence of anomalies of refraction, through the neglect of suitable glasses after the onset of presbyopia; macular disease, hyperæmia of the chorioid with occipital or frontal pain, often occur after forty years of age. The tougher sclerotic coat of adult life is not so liable to distention as in childhood and youth, but the reflex pain, hyperæmia of the conjunctiva and impaired nutrition of the globe are equally imminent, and relief comes only after sufficient time has elapsed, under suitable hygienic conditions, to permit a subsidence of the pathologic conditions of the tissues. In many patients the use of glasses is not sufficient, but careful and persistent therapeutic measures are required, in addition to the rest from work. Time for recovery from the local, secondary results of eye-strain must therefore be allowed in many patients before it can be said positively that the eyes are not responsible for the headache.

The subsidence of pain is not infrequently gradual, the paroxysms being less frequent, not so severe, and of shorter duration, until finally they fail to return, or only at long intervals, and then under special stress of circumstances. The etiologic relation of these ocular conditions to disease of the lining membranes of the bony sinuses of the skull which lie contiguous to the orbit is too frequently overlooked. That sinusitis is a frequent cause of certain forms of headache is well known, but that the eyes participate in the congestion due to blocking up of the exits from these sinuses into the nose has not met with the wide recognition its importance suggests.

It will be observed in the foregoing paragraphs, therefore, that in the study and treatment of ocular neuroses many complex conditions are encountered which require a conservative application of knowledge, skill, and wide experience for their successful management. It is possible that the ophthalmologist may be prone to exaggerate the importance of ocular affections as a factor in producing headache and general nervous symptoms, but this should not cause surprise when an analysis of one thousand private patients, applying consecutively for treatment, revealed the fact that approximately 50 per cent. of these complained of this symptom, and a considerable percentage of these headache patients came by the advice of their physicians and were unconscious of any ocular defect. While his daily experience might therefore lead him to overestimate the significance of eye-strain, it has taught him that there is great danger of its importance being overlooked or ignored by those laboring in other fields of practice.

There can be no doubt as to the fact that exaggerated importance has often been given to minor errors of refraction in patients whose eye symptoms,—*e.g.*, hyperæmia of the fundus, impaired function, headache, undue sensitiveness to light, etc.,—were due to other causes than eye-strain. That these symptoms of eye disease are due to some general dyscrasia,—*e.g.*, lithæmia, diabetes, and general blood-vessel disease,—is well known to all careful observers.

That very small errors of refraction are frequently the cause of headache and other symptoms is true, but other sources of disturbances should be carefully excluded before ascribing the symptoms to eye-strain alone. Many persons with delicately poised nervous systems are nevertheless greatly benefited by the correction of very low degrees of astigmatism, particularly if the slight error of refraction is associated with ocular imbalance, and they are unable to abandon their glasses since any attempt to do so leads promptly to a return of their headache.

In others, and they constitute a large group of persons who wander from one consulting room to another, the writer is convinced that the glasses afford the temporary relief which they claim to experience, by a species of suggestion, precisely as they would be relieved for a short time by the mummeries of a Christian scientist or the manipulations of an osteopath. Many women who have been despoiled of their ovaries belong to this group, and while they are often unable to go without their correcting glasses, never secure entire relief from their headaches, which they associate with any continued use of their eyes.

The fact that many persons, even children, often abandon their glasses after wearing them for a few months, and that without return of their headache, is not to be accepted as proof that their symptoms were in the first place wrongly ascribed to eye-strain. Improved health, a long summer vacation, or change of environment, as for example, a better-lighted room or less pressing work, may enable them to bear the eye-strain with impunity, but the glasses had probably carried them over an important interval and prevented permanent injury.

In treating headache demonstrably due to eye-strain, and for the present contention excluding from consideration all other possible causes and associated conditions, the writer will now pass to the discussion of the methods which experience has taught to be of greatest efficiency.

In patients who suffer but little pain but complain only of blurring page after protracted effort at near-work, and in whom the ophthalmoscope shows a healthy eye ground, it is often sufficient to supply these with so-called manifest corrections; but in the class of persons under discussion in this chapter who, by reason of eye-strain are victims of headache and present the local inflammatory or irritative changes which have been described, it is necessary to use one of the mydriatic alkaloids to temporarily suspend the action of the ciliary muscle, in order to determine with accuracy the static refraction of the eye.

The drugs usually selected for this purpose are the sulphates of atropia, hyoscyamine, and duboisine, and the hydrobromate of homatropine. Others are employed, but those named are more commonly used. These drugs possess not only the power of dilating the pupil and paralyzing the ciliary muscle, but are also local anæsthetics or analgesics. Their value is twofold. In the first place they secure to the eyes a rest from accommodative efforts, and so relieve the eye-strain; at the same time through their soothing influence they aid materially in relieving the nervous irritability of the eye. It has been claimed in the foregoing pages that the immediate result of the accommodative strain constantly required to overcome some anomaly of refraction is to produce hyperæmia and turgescence of the ocular tunics, and that this, together with the exhaustion of the nerve-centres involved, is in great measure the cause of pain in the head and its associate reflex phenomena. The advantages therefore accruing to the patient from the use of the salts of mydriatic alkaloids are obvious. The pain in the eyes and head, the insomnia, the external redness and the turgescence of the uveal tract rapidly subside. These drugs should not, therefore, be regarded merely as cyclophlegics or paralyzers of the accommodation only, but as important and efficient therapeutic agents in the treatment of the ocular conditions associated with headache. To the class of patients under study this contention is of signal importance, since for their relief approximate corrections of the errors of refraction are not sufficient. The exact nature and degree of the defect must be accurately known, and this cannot be determined in a pair of congested, irritable eyes, with a fluffy chorioid, and exhibiting undue sensibility to light. Before any trustworthy data as to the static refraction can be secured, the eye must have time to recover from these conditions, and recovery is not only more rapid under the use of mydriatics than any other known means, but in most cases they are the sole means by which satisfactory results may be secured. This is particularly important where diseased changes at the macula are present.

Many failures to demonstrate ocular responsibility for headache are due to neglect at this point. So-called manifest corrections of refraction errors in this class of patients are valueless for diagnostic purposes. All hurry-up methods are almost equally so. There is much variety in the opinion and practice of different ophthalmic surgeons as to the best methods of employing the cyclophlegics. Many prefer to use the stronger and more persistent members of the group; for example, solutions of the sulphates of atropine or hyoscyamine, while others use almost habitually the hydrobromate of homatropine and consider it sufficient. Homatropine possesses the sole advantage of being evanescent in the duration of its mydriatic and cycloplegic properties. The writer demonstrated its efficiency, in 1881, for the determination of errors of refraction in healthy eyes, but in headache patients the eyes do not as a rule belong to this class. For these patients homatropine is not efficient unless, in strong solution frequently instilled, it is continued day after day until the pathologic conditions of the *fundus oculi* have subsided, in which case it is better to adopt at the outset one of the stronger, more efficient, and enduring drugs.

Then, too, after the measurements have been made, the correcting glasses ordered and the mydriatic omitted, another practical advantage is secured by the long-continued influence of the cycloplegic. The glasses should be worn by the patient as soon as possible so that the power of accommodation shall be recovered under their use.

An important factor in the disturbance of function in all cases of refraction error is the derangement of the binocular balance. That is to say, since the convergence and accommodation are under the same innervation impulse, the existence of any refraction error which increases or diminishes the amount of accommodation required to secure a clear image upon the fovea of each eye, must of necessity disturb the relation of the accommodation impulse to that required for convergence of the two eyes to the same point.

Theoretically, therefore, every hypermetropic or myopic eye would have as an alternative a choice between a dim or imperfectly focussed image upon the fovea, or double images, for the reason that the eyes with a convergence impulse corresponding to the power of accommodation required to be exerted for the clear image, would not be converged to the required point, but in hypermetropia to a too near point and in myopia to one too remote. The fusion sense has, however, taught most persons the trick, in hypermetropia, of accommodating within narrow limits without convergence, and in myopia to converge to the required point without accommodation, a fact pointed out by the investigations of Donders and designated by him as "the relative range and region of accommodation and convergence." By this means the alternative of dim sight or double sight, in most individuals, is in large measure, or entirely, averted. This result of lifelong experience, however, becomes a disturbing factor when correcting glasses are attempted. As soon as the necessity for the accustomed undue strain upon the accommodation is removed by a convex glass, as in the correction of hypermetropia, it becomes necessary for satisfactory binocular vision that they should unlearn the habit of a lifetime and exert the same innervation impulse for both the convergence and accommodation, as in emmetropia. The great difficulty many persons have in learning to wear a pair of correcting glasses grows out of the fact that they cannot adjust themselves to this new order of things immediately. They do so, however, with much less difficulty if the glasses are worn during the days required for the cycloplegic to disappear. The gradually returning accommodation affords an opportunity for a readjustment of the new relations between convergence and accommodation. Having these considerations in view, the glasses for headache patients certainly should be prescribed at once and worn steadily.

The careful study of the binocular balance is a most important phase of the treatment of eye-strain. It will be found, if appropriate means are employed, that a large percentage of patients applying for relief from eye-strain symptoms present some form of ocular imbalance. Esophoria and exophoria, or low degrees of vertical deviation are the rule. Fortunately, under the sufficiently prolonged use of a mydriatic, many of these deviations will be found to have disappeared when sought for through accurately adjusted glasses which correct the existing defect of refraction. There is nevertheless a large percentage of them which remain as absolute defects of balance between the two eyes. Those which disappear are related to the muscular strain and anomalous innervation impulses brought about by the attempt to overcome the defects of refraction in the interests of clear vision. I have elsewhere designated them as relative heterophorias. When the strain is removed by glasses and clear vision secured without undue accommodation effort the tendency of the imbalance is to disappear, either spontaneously, or by suitable muscular exercise with prisms.

The permanent defects of balance have, however, an anatomical basis, as already pointed out (see page 6) and require careful analysis and separate treatment.

The following clinical history will serve to illustrate a somewhat numerous class of patients with defective vision who apply for relief from their painful eyes, headache, and a complex of symptoms which can be regarded only as ocular neuroses.

Mr. K., aged 26, applied for treatment in December, 1899. He was wearing the following glasses ordered by Saemisch of Bonn, in 1894:

O.D. $-\ 2.75^{c}$ ax. 105°
O.S. $-\ 1.75^{c}$ ax. 90°

with these V.=O.D. 6/60, O.S. 6/9. He suffered from constant fronto-occipital headache which frequently culminated in violent attacks of pain and general "nervousness" which drove him from business and to bed. He carried his head tilted to the right; the eyes were red, unduly sensitive to light, and he suffered from blepharitis and recurring styes. The ophthalmoscope showed a fluffy, turgid fundus in both eyes, and in the right a distorted disc, and a large conus below, with concentric, crescentic markings indicating successive fresh encroachments upon the chorioid. The axis of the conus was at 120°. In the left eye the conus was at the temporal border of the nerve and beyond it; approaching the fovea there were granular changes in the chorioid. The right eye was on a plane in front of the left, the right side of the face was larger than the left, the zygomatic arch being prominent, while on the left it was flat or slightly concave. The left eye was nearer the bridge of the nose than the right. The ophthalmometer indicated in the right eye a corneal astigmatism of 4.D., the principal meridians being at 30° and 120° respectively. In the left eye an astigmatism of 3.D. the meridians lying at 170° and 80°. After prolonged mydriasis and rest a careful measurement by objective and subjective methods resulted as follows:

$$\text{O.D.} - .25^{s} \subset -4.00\text{ D. cyl. ax. } 30^\circ \text{ V.} = \frac{6}{\text{VII}\frac{1}{2}}.$$
$$\text{O.S.} - 1.25\text{D.} \subset +2.50\text{ cyl. ax. } 80^\circ \text{ V.} = \frac{6}{\text{VI.}}$$

Through these correcting glasses, carefully centred, the ocular imbalance was measured at 6^{m}. and revealed a left hyperphoria of 8° and an esophoria of 6°. These deviations would vary, the hyperphoria becoming greater, while the esophoria diminished, or the reverse would occur, in the meantime the upper end of the vertical meridian of each cornea rotating outward (cyclophoria). The sketch on page 747, Fig. 1*a*, taken by his hatter, gives the skull outline. The septum, in the nose, was deflected toward the left side, and the left middle turbinate was firmly wedged between it and the bony wall of the nostril. The roof of the mouth was a deep cathedral arch. This case presents a somewhat exaggerated picture of the anatomical basis for many cases of anomaly of refraction associated with ocular imbalance. His father and mother and a younger sister were subjects of hypermetropic astigmatism, and headache was a family trait. The eyes of this young man were evidently distending, the right having passed into myopic refraction, and the left was myopic in the vertical meridian, the horizontal being still hypermetropic, i.e., mixed astigmatism. The measurement and correction by glasses of the refraction error for each eye was not difficult, but the obvious difficulties presented in any effort to secure comfortable binocular vision illustrates forcibly the complex problem often presented in these cases of ocular imbalance.

It is by no means always an easy task to discern the precise nature of the muscular defect, or which of the extra-ocular muscles is at fault, or whether the muscle or muscles at fault are so by virtue of impaired innervation, faulty development, or as the result of abnormal attachment to the globe. The analysis of the ocular movements, however, is often greatly facilitated by making the study at 6^m, while the accommodation is still paralyzed. For this purpose it is important to employ some fixed form of apparatus in which the lenses required to correct the existing refraction error for each eye can be placed and carefully centred. The examination should be made with the eyes in the primary position, using a small point of light placed at six metres from the patient, preferably before a black background, from which all objects which may distract the attention are removed.

Much skill, patience, and experience are required in the diagnosis and treatment of these cases of imbalance due to absolute muscular defects. In some instances, when there is much distortion of the walls of the orbit and of the peri-orbital region of the skull, it is probable that no amount of skill either by surgical interference or by other means can bring about comfortable binocular vision.

One of these patients after many operations at the hands of several men of wide reputation came to the writer with the request that he should remove one of her eyes as the only means of promising any hope of relief from her distressing headache and nervous symptoms. The request was refused, but was subsequently granted by an eminent surgeon in another city. It is probable that repeated tenotomies, advancement of tendons, etc., to which her eyes had been subjected had done harm, certainly had failed to afford relief. It is true, nevertheless, that there is a large number of patients who do not secure relief from accurate correcting glasses, whose continued trouble is unquestionably occasioned by a defective balance between the eyes which is susceptible of correction by surgical means. The power of convergence is insufficient or it is relatively too great for the power of abduction, or in the position of rest one eye is above the horizontal plane, or the other below it, and so binocular single vision is either intermittent or impossible, or is maintained by virtue of constant muscular tension which causes headache, etc. Experience has shown that in the minor degrees of these faulty muscular conditions, the suitable use of prisms, combined with the glasses required for the correction of any existing error of refraction, often give practically complete relief. In the higher degrees, however, surgical interference, designed to restore ocular balance, is theoretically a correct procedure and its value has been abundantly justified by results. Where failure is met it may often be ascribed to mistaken diagnosis, usually as to the nature of the fault the surgeon has sought to correct, or possibly to a faulty technique, or to some unavoidable accident,—*e.g.*, excessive hemorrhage into the capsule of Tenon, or to excessive inflammatory reaction during repair. In two cases seen in consultation, advancement operations had led to overcorrection of the defect through the contraction of the resulting cicatricial tissue. Mistakes may occur in the selection of the wrong muscle for surgical procedure. For example, tenotomy of an apparently too strong muscle may be performed when advancement of the weak one should have been the operation of selection, or, the so-called secondary deviations may mislead the incautious operator. Then, too, it is not always easy to differentiate between deviations due to faulty attachments of a muscle tendon or a partial palsy of a muscle.

It is not the intention in this chapter to present a treatise on either errors of refraction or the heterophorias, but to show the responsibility of these ocular abnormalities in causing ocular neuroses. In the presence of these defects of vision the associated headaches should be regarded as possibly due to them until they have been corrected, and the resulting local pathologic states have been cured.

Unfortunately, but little is actually known of the real mechanism of headache. For our present purpose, however, it is fortunate that the mechanism of eye-strain, if not of the resulting pain, is well understood. It is probable that no analogous conditions are presented elsewhere in the body. An illustration, however, of the possible results of eye-strain is afforded by writer's cramp. In the hypermetropic eye, for example, clear vision is maintained only by the constant contraction of the ciliary muscle. When astigmatism is also present, or if the defect of refraction is heteronymous, or of differing degrees between the eyes, or if the tormenting effect of ocular imbalance is added to the problem of maintaining clear vision, the difficulties of the struggle are greatly enhanced and the resulting exhaustion or derangement of the nerve-centres is more rapid and enduring. No muscle or group of muscles in the body could endure with impunity the exhausting fatigue to which the ciliary muscles and their associated group of extra-ocular muscles are subjected, by the above described conditions, for the hours ordinarily required for the purpose of vision. To this muscular fatigue must be added the exhaustion of the nerve-centres involved in the innervation of the ocular muscular apparatus, the resulting congestion of the intra-ocular tunics and the consequent pressure upon the terminal distribution of the ciliary nerves, and possibly some subtle derangement of their nutrition occasioned by the impaired circulation produced by the increased intra-ocular tension. It is plain that in neurasthenics or feeble individuals, or in industrious people who use their eyes persistently under the added strain of near-work, the exhaustion would be relatively more complete and enduring than in vigorous and relatively indolent individuals. While the latter might endure with comparative impunity the imposed strain, the former groups of individuals would be forced into semi-invalidism.

PART II.

BY HOWARD F. HANSELL, A.M., M. D.

GENERAL CONSIDERATIONS: EPILEPSY; CHOREA; VERTIGO; GASTRIC DISTURBANCES; SPASMODIC MOVEMENTS OF THE HEAD OTHER THAN CHOREIFORM; GENERAL ASTHENOPIA.

GENERAL CONSIDERATIONS.

THE tendency of modern medicine is encouragingly strong in its insistence that the diagnosis and treatment of disease shall be reduced to a demonstrable basis. Dogmatism and empiricism have been pushed farther and farther into the background of recent thought and literature by the revelations of the microscope, the exactness of physiologic research, and by the accuracy of trained work and observation in the laboratory. Disease is not general to the system, but special to some part of it. It has a local, if not a discoverable, habitation in the human economy, and a cause which it is the mission of science to discover and remove. The more general recognition of this fact is bearing fruit in the minds and labors of medical men. Each year fewer diseases are classified by their symptoms, and more are referred to the category indicated by their etiology. As the causes of disease are laid bare, prophylaxis and treatment are simplified and become more efficacious. Surgery has broadened to the inclusion of affections that were considered, not many years ago, to belong strictly to medicine, while the specialists are invading the old fields of both medicine and surgery. They are rightly accorded a more and more extended domain as their importance grows in recognition. This is particularly true of ophthalmology. The well-directed and resourceful labors of earlier workers in this specialty, notably Thomson, Weir Mitchell, Dyer, Norris, Harlan, and younger men, trained in their clinics and influenced by their teachings, have forced the acknowledgment of ophthalmology as an indispensable adjunct to the practice of medicine and surgery. Its services to the medical sciences is especially great in the diagnosis and treatment of the so-called functional diseases of the nervous system, and organic affections of the brain and abdominal organs. This specialty is also a brilliant contributor to the diagnosis of obscure muscles of convergence and divergence. The primary cause—hyperopia—thus initiates a series of morbid phenomena, abnormal muscular action and centrifugal impulse that eventuates in a deranged ocular function which, if not corrected, involves the general health and nervous organization, exhibited by definite, pronounced, vague, or bizarre reflexes. The eye-strain in myopia, is, conversely, due to the unassociated action of convergence and accommodation, the tendency to monocular vision and the intense effort to converge and maintain binocular singular vision for all distances. The eye-strain in astigmatism is that of hyperopia or myopia in conjunction with the unequal contraction, hence innervation of the ciliary muscle in the constant effort to obtain clearly defined retinal images. (See Chapter II.)

The ill effects of the use of the eyes in all forms of ametropia are referable to the eyes as well as to the muscles which control their movements. In most cases an error of refraction is the primary cause, and muscular anomalies are secondary and dependent affections. In a small minority of cases, however, as in anatomical peculiarities of muscular insertion, malformations of the bones of the orbit or skull, or in muscular weakness or paralysis, eye-strain may exist as the result primarily of muscular incoördination. These conditions, irrespective of the refraction of the eyes, react upon the central nervous system and occasion manifestations of disordered function exactly as though the muscular anomaly were secondary to ametropia.

2. The symptoms of uncorrected presbyopia, or the gradual failure of accommodation in advancing years, are indistinct near vision, recession of the reading distance, the need of stronger light and larger type, headache, and other forms of asthenopia. To these increasing disabilities may be added, in exceptional cases, any or all forms of ocular reflexes common to ametropia and muscular imbalance. The direct and indirect symptoms are referable to the strain of an accommodative power weakened by lowered contractility of the ciliary muscle as a part of the general muscular loss of tone coincident with age, and to a gradual stiffening and loss of elasticity of the crystalline lens. Such an eye is no longer able to adapt itself quickly, if at all, to the demands of vision at close range, and the nerve stimulus to the ciliary muscle, probably not lessened by age, meets with insufficient response. Hence, vision is blurred and cannot be cleared except by the employment of convex lenses.

3. There is an interdependence of the functions of the intraocular and extraocular muscles. Every accomplished act of accommodation, and every rotation of the eyes in any direction, is consummated by the

ocular muscles; and in (4) the connection of the peripheral organs of vision with the central and sympathetic nervous systems.

1. Emmetropia and equilibrium of the ocular muscles present ideal conditions. The emmetrope receives on his retina clear, defined images of distant objects (6 m. or more), with minimum effort of accommodation and contraction of the extraocular muscles (equilibrium and binocular fixation). For objects nearer than infinity and to within 33 cm. (the reading distance) of the eyes, the refracting power of each eye is increased by the uniform, gradual or sudden contraction of the ciliary muscles and the simultaneous contraction of the muscles of convergence, the equivalent relaxation of the muscles of divergence and the maintenance of the same visual horizontal plane for both eyes by equal tension of the muscles of supraduction and infraduction.

That equilibrium and physiologic relation between the action of intraocular and extraocular muscles shall be originated and sustained, nerve stimulation and muscular response must be normal. Such a combination implies a healthy condition of the centres of vision, an uninterrupted and unimpaired function of the efferent and afferent nerves and that their peripheral distribution is normal. The separate muscles of both eyes do not co-ordinate perfectly unless they have normal relations with each other and with the eyeballs. The development of ocular reflex neuroses in emmetropia and extraocular muscle balance must be referred to an extraocular cause, such as the weakened or deficient action of the central nervous system, or of the circulatory apparatus, as in convalescence from exhausting disease or confinement, affections of the organs of the chest or abdominal cavity, prolonged near use of the eyes among unfavorable surroundings, the gouty or rheumatic diathesis, venereal excess, overindulgence in stimulants or narcotics, or other source by which the visual apparatus may be deranged. The remedy is not far to seek. The restoration to health of the diseased organs, the avoidance of all excesses, and the use of the eyes under favorable conditions and for a reasonable length of time, will be followed by a gradual return to normal condition.

In the presence of an error of refraction, the physiologic relations between the peripheral organ of vision, including its extrinsic muscles and the cerebral centres, are disturbed. For example, in hyperopia abnormal contraction of the ciliary muscle is required for clear vision. Thus the muscles of convergence receive an excess of stimulation, those of divergence a stimulation so modified that they may yield to the demand of convergence, and those of elevation and depression a degree of innervation corresponding to their anatomical relations with the [illegible] affairs of business and professional life [illegible] the opportunity [illegible] the individual to preserve [illegible] endurance almost [illegible] the end of [illegible] that in our strenuous American [illegible] general break-down of the system.

In [illegible] comparatively insignificant muscular [illegible] more sanitary conditions [illegible] are provocative of ocular [illegible] a serious and more widely disseminated nature.

An editorial [illegible] *Medicine*, February 20, 1904, states [illegible]: "One Overlooked Factor in the Increase of Insanity. [illegible] in modern civilization which is not recognized [illegible] is known to be a profound source of many derangements [illegible] reading. The compulsory or chosen occupation of reading and near use of the eyes in a thousand occupations has [illegible] the life of the city dweller. Printing, [illegible] cheap books, newspapers, and handicraft occupations have appeared and [illegible] a physiologic or unphysiologic activity, whether harmful or not, is a new function, and one to which in evolution neither the mind nor the body has been habited, and especially [illegible] most delicate and most used sense organ [illegible]. Moreover, this sense organ is demonstrably imperfect [illegible] proportion of all persons, and the action of the malformed eye, as has been demonstrated by science, and by thousands of clinical cases, begets disturbances of eye, of cerebral action, of feelings, and of nutrition. Malfunction is the beginning of all disease, and of many it is the middle and end. Suicide, it has been found, is exactly in proportion to the number of hours of school life and study demanded in the country concerned. To this suggestive fact is now added the demonstration that insanity is almost precisely in the same proportion. When, lastly, it is observed that the use of the eye demanded by the civilization of the last century or two is one that its evolution has never provided for, either in structure or in physiology, one gets a glimpse of morbid mental action in the making. When it is found that the insane and criminal have an astonishingly high proportion of high optical defects, the suggestion grows to conviction that the morbid action of the eye and its resultant cerebral functions are considerable causes of the increase of insanity."

But the eyes are not peculiar as disturbing agents of the human body. Physical weakness in one part of the body interferes with the functions of the entire organization. Moreover, asthenopia, whether refractive or muscular, reacts upon the weakened body, and aggravates the symptoms of nervous exhaustion by still further reducing assimilation of food and constructive metamorphosis. In this sense, eye-strain is a cause as well as an effect. In health, on the other hand, ocular defects of considerable degree are suppressed, have no deleterious effects upon the nervous system, and are recognized only by a lowering of the visual acuity from failure of accommodation or from appreciable loss of muscular co-ordination.

Eye-strain, productive of local symptoms and reflex neuroses, is thus accommodative and muscular, the former referring to overaction, and the latter to imbalance, or unequal tension of the muscles which govern the eyes in their rotations in the orbital cavities. Manifestly, one-eyed individuals are excluded from the second class. This fact applies to those who literally have but one eye, but also to persons who do not, under any circumstances, have binocular single vision. Muscular asthenopia is also absent in paralytic or functional squint. This is obviously true, as the essential factor in the causation of ocular pain or distress is the maintenance of binocular fixation under the stress of muscular imbalance of a degree not incompatible with the simultaneous use of both eyes. The indirect cause of asthenopia is the tendency to turn the visual axes away from parallelism; the direct cause, the tension of the opposing set of muscles, whose function it is to resist the tendency. For instance, esophoria, the tendency to cross the visual axes, creates an abnormal effort on the part of the abducting muscles in order to preserve binocular single vision, and it is this extra innervation, and extra muscular response, which is the source of the distress. And this is true whether the muscles of convergence are relatively strong or those of divergence relatively weak.

As already stated, errors of refraction and muscular anomalies are the ocular causes of accommodative asthenopia; of the former, hyperopia and hyperopic astigmatism are of the most frequent occurrence; of the latter, hyperphoria is most common. Among the general or constitutional causes may be mentioned neurasthenia, heredity, nervous affections, debility of any nature, sexual excess, gout, rheumatism, or any condition of the system in which the power of endurance is reduced below the healthy standard. Frequent contributory causes are constant application of the eyes in close work, prolonged reading of fine type, too much or too little light, fine sewing and embroidery, and reading in moving vehicles, or with the head in a position either on a line with or below the level of the trunk.

Inasmuch as a pure horizontal or vertical tendency of the visual axes may not exist [excepting in rare abnormal tensions and relaxations originating in anatomical abnormalities, as a separate and distinct condition, uncomplicated with an upward (in predominating lateral tendency) or a horizontal (in vertical) tendency], we are forced to deal with an obliquely directed tendency to deviation of the visual lines. A consideration of the heterophorias as being the dominant causes of asthenopia and reflex neuroses, necessarily involves a study of the relative strengths of adduction, abduction, and sursumduction for objects at all distances. If in emmetropia adduction equals, approximately, 24° and abduction 2°, esophoria would be due to insufficiency of the muscles of divergence. If right (supraduction) = 5° (left infraduction) and left supraduction = 3° (right infraduction) the elevators of the right eye or the depressors of the left eye would be at fault. If right supraduction (left infraduction) = 3° and left supraduction (right infraduction) = 1° the fault would lie with the elevators of the left eye, or depressors of the right. For measuring the limits of rotation of the cornea in the horizontal and vertical meridians, the clinoscope of Stevens is valuable. By its reading, in degrees of the circle, the excursions of the eye may be readily compared. In addition to the alternate vertical tendency already mentioned, cases are met with in which the vertical inclinations are present and of the same direction in both eyes. Anaphoria is the name given to designate upward, and kataphoria downward, abnormal tensions of both eyes. Such conditions are the result of pathologic states of the muscles or nerves supplying them, or of some obscure unknown disease. They are not satisfactorily explained by errors of refraction or by the muscular contractions induced by the effort to see clearly. It is of vital importance to know that such conditions exist, and that they may cause eye-strain and reflex anomalies.

EPILEPSY.

There is no uniformity among authors in their definition of epilepsy. It is hardly possible to define it exactly. Epilepsy is a symptom expressive of an organic or functional derangement of the nervous system. It is not a disease *per se,* etiologically or pathologically, but a symptom complex. One of the tersest, most accurate, and comprehensive descriptions of the commonly accepted meanings of the word is given negatively in Gower's "Diseases of the Nervous System," published in 1888. "The term epilepsy," he writes (page 1078), "is applied to a disease in which there are convulsions of a certain type, or sudden loss or impairment of consciousness, but in which the convulsions are not directly due to active organic brain disease, to reflex irritation, or to abnormal blood states, and in which the loss of consciousness is not due to primary failure of the heart's action." Subsequent writers have not improved this definition. H. C. Wood defines epilepsy[1] as "A disease of unknown pathology in which, at irregular intervals and without obvious existing causes, an abnormal disturbance of nerve force occurs, in most cases accompanied with loss of consciousness, and very frequently with convulsive disturbance." Landon Carter Gray[2] admits that "much confusion has been injected into the subject by the failure to recognize that epilepsy is only a symptom, just as is fever or cough, and that in every case, the question of diagnosis is resolved into the further one of what the epilepsy is a symptom of." Other authors might be quoted without adding useful evidence to the universally admitted opinion that the so-called disease, epilepsy, is the accompaniment of not one but many affections, in some of which organic changes in the nervous system are found after death, while in others failure to recognize that any of the tissue is diseased is the only result of microscopic examination.

Epilepsy, as a symptom of disease, has no pathology. Authorities are conflicting and inconsistent as to this question. It is, for instance, classified as a functional neurosis, and yet pages are devoted to the discussion of its pathology. Local and general convulsions are due, in many cases, to organic changes in the brain or spinal cord, and are unscientifically classified as functional affections, when they should be designated, for example, as "epileptic convulsions in hemiplegia." Exactly identical convulsions may appear as reflex symptoms of some peripheral lesion. A wider recognition of these facts would lead to a more tenable classification of epilepsy than that usually given, and would tend eventually to eliminate such objectionable adjectives as idiopathic. The subdivision of the subject would then be "Epilepsy from organic disease of the nervous system" and "Epilepsy from peripheral irritation," although the exciting cause might remain unknown in many cases.

The testimony of too many trustworthy clinicians has been published in confirmation of the statement that epileptic convulsions may be due to affections of other organs than the brain and spinal cord, to permit the causative relation of distant lesions to the development of the affection to be ignored. It must be admitted, however, that some other factor, aside from the peripheral lesion, must be active. In considering the nature of the factor, we must assume that some state or condition of the nervous system makes it peculiarly receptive, impressionable, and responsive. And the question arises, What are the predisposing and underlying causes of epilepsy? We confess our ignorance here, as we are forced to confess it in many other affections, and cover our lack of exact knowledge by the use and abuse of such names as [illegible], susceptibility, idiosyncrasy. We cannot explain by the results of pathologic examination or post-mortem studies why an old cicatrix should cause epileptic attacks in one individual and not another, or why intestinal worms will cause corneal ulcer in one child and not in all children, without having recourse to the intangible but none the less cogent influence of personal susceptibility, an indefinable quality, inherent and peculiar to the individual.

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[1] Pepper's System, Theory, and Practice of Medicine, 1893.

[2] Nervous Diseases, by American Authors, edited by F. X. Dercum, 1895.

Etiology. In some respects the theories of the diagnosis and treatment of functional neurosis need reconstruction. It can certainly not be claimed by any enlightened medical man, however earnest and enthusiastic he may be, that all or even a large proportion of "idiopathic" cases are the reflex result of perverted function of some distant organ. On the other hand, in view of the authoritative statements and experience of the general practitioner, it cannot be doubted that at least a minority of such affections are reflex in origin. H. C. Wood[3] divides epilepsy into organic, toxæmic, Jacksonian, idiopathic, and reflex, and gives as causes, wounds of the head or other portion of the body, astigmatism or other imperfection of the eyes, diseases and malformations of the nasal cavity, carious teeth, retained milk teeth, aural diseases, adherent prepuce or other irritation of the genital organs, and intestinal worms." W. B. Pritchard[4] gives as causes of reflex epilepsy, the existence of which, he says, is somewhat widely questioned, phimosis, adherent prepuce or clitoris, stenosis of uterine os, nerve cicatrices, or surgical disease of the limbs or joints implicating nerve trunks, carious teeth, eye strain (esophoria, exophoria), obstructive diseases of the nasal passages, aural disease, etc., etc. Herter and Smith,[5] in considering the etiology of epilepsy, mention as one of the most important causes "putrefactive changes in the intestinal contents, toxic substances produced in the intestines" (reflex epilepsy). Stoewer[6] says

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[3] Pepper's Text Book.

[4] Sajous's Annual, vol. iii., 1899.

[5] New York Medical Journal, August and September, 1892.

[6] Augenkrankheiten als Ursache der Epilepsie, Klin. Monatsb. f. Augenh., 36, 1896.

"most cases are laid to the cerebrum, while for other cases, certain irritations of peripheral nerves are necessary for outbreaks of an attack," and "many of the last reflex class are cured by the removal of painful scars, irritation from nose and ear." P. F. Barbour[7] reports the case of a male, married, who had epileptic seizures since 1892. He found a marked increase of indican in the urine and dilatation of the stomach. Hence he inferred that the trouble arose from intestinal indigestion and constipation. Treatment for these conditions relieved the epilepsy.

These quotations and abstracts, and more might readily be added, are sufficient to prove that epileptic attacks may be reflex in origin, and that removal of the source of irritation will re-establish normal function of the nervous system. It is, therefore, logical to assume, and the assumption has been abundantly verified, that reflex epilepsy may be the result of eye-strain, accommodative and muscular. The relation of the function of the ciliary muscles to the extraocular muscles, has already been alluded to. Most cases of accommodative asthenopia are also cases of muscular asthenopia. Therefore, both causes may be active in the same individual, and should be taken into account as factors. The relative importance, in the etiology of nervous affections, of refraction and muscular anomalies, is difficult to determine. Some authors ascribe the whole responsibility to refractive errors, others to want of equilibrium of the extraocular muscles, and not a few writers utterly ignore the eye and its muscles. Strohmayer,[8] for instance, dips deeply into the consideration of etiology and treatment by medical and hygienic measures. The omission in a systematic monograph of such recent date is surprising, and the only inference, of course, is that the writer does not believe in eye-strain as a cause of epilepsy. Landon Carter Gray[9] makes a distinction between the two sources of eye-strain. "The removal of insufficiencies of the ocular muscles is, in my opinion, quite useless, but marked errors of refraction should be remedied." The reasonableness of this decision, half-hearted though it is, may well be questioned. The dual cause is so intimately and so inseparably bound together, that a separation or elimination of either is not only unscientific but inconsistent with intelligent criticism.

M. Allen Starr[10] believes that epilepsy is an organic disease. He bases his opinion on the study of 4000 cases and his researches are certainly worthy of careful attention. One hundred and ninety-two of the 4000 cases exhibited positive signs of non-development of the brain, 68 per cent., signs of epilepsy before twenty years of age, the period of brain development. The most frequent cause was heredity, and the next in frequency, trauma of the head. That the latter was a more potent cause than wounds of other parts of the body, was well shown by the statistics of the Franco-Prussian war. These were conclusive. Out of 8889 soldiers wounded in the head, 46 became epileptic; whereas, out of 77,463 persons wounded elsewhere, only 17 became epileptic. Fright was undoubtedly a cause, as 119 of the patients tabulated had the first fit after a fright.

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[7] Medical Times, February, 1895.

[8] Die Epilepsie im Kindesalter, 1892.

[9] Nervous Diseases, by American Authors.

[10] New York Medical Journal and Philadelphia Medical Journal, January 30, 1904.

C. L. Dana, in discussing Starr's paper, said, "At least ten years ago he had spoken of epilepsy as a progressive degenerative disease of the brain, but, as we did not yet know always what this degeneration was or where it was, and could not give a morphological description of the diseased cells, it was probably wise still to describe epilepsy as a disease without definite organic basis. He also doubted if the term disease was the proper one to use in connection with epilepsy. There was certainly an underlying organic degeneration, but it seemed to him that in many cases it was rather a morbid condition than a pathologic process, rather a teratological defect than an actual disease. So far as we know, it was probable that the most important factor underlying epilepsy was the tendency of large groups of nerve-cells to undergo a rather progressive degenerative process. With regard to the incurability of epilepsy, he expressed the opinion that the stand taken by Starr was not only incorrect but discouraging. He absolutely knew of cases in which epilepsy had been cured. He knew personally of a young man of seventeen, who had nocturnal epilepsy for one or two years, the attacks coming as often as two or three times a month. He had seen the attacks himself and they were characteristic. Under the bromide treatment this man recovered and was now forty-five years of age, and had never had an attack since completing the treatment. There was now none of the interconvulsive symptoms of epilepsy, and this disease should be looked upon as cured, just as much as pneumonia should be considered cured. The very theory of the disease, assuming it to be organic, would make a cure possible. We know perfectly well that there are certain degenerative processes that we may arrest; for example, tabes and degenerative processes affecting the kidney or liver, for degenerative processes were not always progressive." W. H. Thomson, in the discussion, said that he considered functional nervous diseases as true derangements of the source of nerve function, independently altogether of the structure of the brain,—*i.e.,* the blood. Functional disease might be so serious as to terminate life, and yet no nerve cell or fibre could be demonstrated to be diseased, the disorder, perhaps, having been altogether due to a toxin in the blood. He could not believe that a disease so strikingly characterized by intermittency could consist essentially in a structural lesion, nutritive or otherwise. He fully agreed with Dr. Starr that epilepsy was not curable by the surgeon, and stated on the contrary that a surgical operation on the brain was apt to be followed by epilepsy. A polypus of the nose sometimes gave rise to an intermittent asthma. He had had a number of cases in which the removal of peripheral irritation had been followed by the permanent disappearance of epilepsy. With regard to the pathology of epilepsy, we were often misled in regarding epilepsy as due to a discharge in the cerebral cortex. His definition of epilepsy was a "sudden derangement of the normal inhibition of cortical centres upon one another, dependent in the first instance upon an abnormal afferent impression." R. H. MacAllister[11] analyzed 250 cases of epilepsy treated in C. L. Dana's clinic and private practice, and does not mention ocular affection among the etiological factors.

Published cases of the cure of epilepsy by the removal of peripheral irritations are not uncommon, and include many varieties of lesions. Pecho[12] saw patients recover after enucleation of an atrophied ball, following an injury in early life. Galeowski[13] records the history of a man, aged forty, whose right eye was injured six years before, and for which abscission of the anterior part of the eye had been performed. The stump of the right eye was still very sensitive. For six months before he came under the care of Galezowski he had frequent epileptic seizures, and vision in the left eye had been failing. The ophthalmoscope showed left neuroretinitis, venous congestion, perivascular infiltration, and retinal hemorrhages. The stump of the right was sensitive to pressure. After enucleation of the stump, the epileptic attacks disappeared and vision in the left eye improved. Stoewer, reported the case of a married, healthy man thirty-two years old, who had been all his life myopic. In 1891 influenza had been followed by an intractable iritis and that in turn by epileptic seizures every few days, headache and eyeache. The eye was blind from closure of the pupil. He had tonic and clonic spasms, in which he became unconscious, and after recovery was extremely forgetful. He was treated by suggestion, electricity, and bromides, without benefit. On the contrary, the attacks became more frequent and violent. The patient became exhausted and emaciated. The attacks immediately ceased, and improvement in health marked, after enucleation of the left eye. Knapp[14] says, "There is a class of cases, however, in which the reflex origin seems fairly probable,—the cases of so-called epilepsy. A critical examination of the cases reported under this head would eliminate many of them. A considerable number of them are undoubtedly hysteria, and in others the dependence of epilepsy upon the alleged source of irritation is certainly doubtful. Nevertheless, the theory that a remote irritation may give rise to convulsive seizures is supported by the well-known experiments of Brown-Séquard, who produced epilepsy in the guinea-pig by injury of the sciatic, and by a limited number of authentic cases in man. In most cases cited, the source of the epilepsy has been of traumatic origin, and upon removal of the source of irritation, the epilepsy has ceased. These cases are, however, extremely rare."

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[11] New York Medical Journal, January 29, 1894.

[12] Rec. d'Ophthal., January, 1886.

[13] Rev. d'Ophthal., January, 1886.

That heredity is an important predisposing factor is shown by Hare,[15] who found, after examination of many cases of epilepsy, that in 25 per cent. a distinctly neurotic family history could be traced.

The above extracts are sufficiently numerous and trustworthy to prove that the symptom complex known as epilepsy may be caused by peripheral lesions in persons who have neurotic tendencies.

Science has been, as yet, unable to discover the post-mortem lesions, or conditions which explain the symptoms of epilepsy in the so-called functional forms. If it were possible to distinguish between functional and organic epilepsies, the whole subject would be elucidated. And yet, no description of epilepsy has been considered complete without a detailed account of the post-mortem findings. The pathology of epilepsy is that of the disease which causes the convulsions and is, in many cases, unknown. Gowers well says that "the changes in the nerve-centres are probably of that kind which is revealed only by altered function and eludes the most minute search. In the absence of any help from anatomy and histology, the pathology of idiopathic epilepsy is a matter of hypothesis." With the organic form of the affection we have nothing to do in this paper, and confine our observations to functional neuroses as indications of peripheral irritation. In such affections there is primarily no pathology; secondarily, as the result of the habit of convulsions, associated with unconsciousness, the cortex may undergo changes which later develop into appreciable anatomical lesions.

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[14] American Journal of the Medical Sciences, October, 1895.

[15] Quoted by Wood in Pepper's Text-Book.

Although the vasomotor theory of the causation of the nerve explosions has been given up as explanatory of epilepsy, it is still more applicable than any other theory to explain the condition of reflex epilepsy. The influence of the vasomotor nerves, originating or passing through the ganglia of the cervical sympathetic, is not thoroughly understood. The effect of exsection of the superior ganglion upon the ocular tissues, however, is sufficiently pronounced to warrant the inference that the nerve impulse that passes out of the ganglion to the cerebral vessels, is modified by the condition of the ganglion, whether diseased or healthy. It may be here incidentally stated that in seeking to find a cure for idiopathic epilepsy surgeons have removed the ganglia in order to note the effect of its absence upon the calibre of the cerebral blood-vessels. The results of sympathectomy upon the eye are thus summarized by De Schweinitz:[16] "Myosis, narrowing of the palpebral aperture, projection of the nictitating membrane, diminished intraocular tension, certain ophthalmoscopic and microscopic lesions in the eye-ground, and possibly trophic disturbances."

The causative relations of derangements of the ocular apparatus to epilepsy have been the subject of no little controversy among neurologists and ophthalmologists. Since the publication of Stevens's prize essay, and the investigation of Stevens's claims by the New York Neurological Society, the attention of the profession has been directed toward eye-strain as a cause of epilepsy, the relief of the convulsions by correction of optical errors, the restoration of balance of the extraocular muscles, and of the normal relationship between the intraocular and the extraocular muscles. The value of Stevens's labors has not been fully appreciated by the general practitioner, or even by the neurologist, and the ophthalmologist's efforts in this field are limited, except as employed in conjunction with his colleagues. The same idea is more forcibly expressed by R. H. Gibbons:[17] "I know a young man cured of epilepsy in a bad form by Ranney, and I know of four cases cured by Stevens. None of these cases has been reported and one of them has been cured for five years. Stevens and Ranney have been misquoted, or worse,—they have been misjudged by ophthalmologists in general and by neurologists in particular." The laity has already learned by experience to apply to the oculist for relief of headache, but is not sufficiently advanced to appreciate that other neuroses may originate in eye-strain. The general practitioner must take the initiative, and, in the treatment of epilepsy, should endeavor to discover and probe every possible cause, including the ocular apparatus, and not wait until all the text-books on etiology have been exhausted before turning the eyes in the right direction. The writer has natural hesitation in expressing himself in a definite, practical manner as to the relation that eye-strain bears to the causation of epilepsy. Evidence culled from books and monographs is conflicting. It must be admitted that advocates of this theory of causation have not been successful in carrying conviction to the minds of men who have had most to do with epileptics. The theory has undoubted claims to careful and impartial consideration, and the work done along these lines is of sufficient weight to compel belief or refutation. The writer's own belief is, that a small proportion of cases of epilepsy, and of epileptiform convulsions, has been cured by eye treatment alone, and that the proportion equals, in percentage of cures, that of any other single surgical or mechanical means. He strongly advocates, moreover, a thorough and systematic ocular examination in all patients in whom the etiology of the convulsions is unknown. In forming this opinion, he is guided, not by his own experience, which is most limited, but by the cases and histories reported in recent literature. Stevens [18] examined the ocular conditions in 140 cases of epilepsy. "The general result has been to reveal the existence, in these cases, of refraction anomalies in a considerably greater proportion than has been found by Cohen in his examination of the eyes of school children, or by other observers in similar investigations prosecuted in Germany, Russia, and America. In 100 consecutive cases, hyperopia was present in 59, myopia 23, emmetropia, or refraction less than 1 D., 18. Twenty-nine cases were treated only by the removal of optical defects. Of these, 14 may be considered well, 2 (still under observation) are believed to be permanently relieved; 3, still under treatment, have received such marked relief that it is believed an entire discontinuance of the malady may be expected. One died, 7 received temporary relief, and 2 manifested no improvement. Some remarkable histories and striking photographs are appended to the histories of a few of the most pronounced cases. Ranney's summary of 20 cases [19] is as follows: Four abandoned treatment. Of the 22 remaining cases, 10, or 45 per cent., may be considered as well; 7 being completely cured and 3 being practically cured; amelioration of the attacks has been afforded by eye treatment in 9 cases, or nearly 42 per cent.; no improvement has been observed in 3 cases, about 13 per cent. Ranney's collection of case histories is truly a remarkable one, and, viewed from an unprejudiced stand-point, convincing. As he says in the general *résumé,* "I have brought forward here some very strong written testimony from physicians of repute, and the patients themselves, to prove that the histories published by me are those of genuine epileptics, that the results were as I stated, and that benefit followed the relief of eye-strain after a failure of medicines or diet to control the seizures." J. E. Colburn [20] examined 43 cases of well-marked epilepsy, and from this number selected 14 which presented subjective and objective symptoms of eye-strain. Those rejected, 29 in number, presented other causes for the epilepsy or failed to afford evidence that the eyes, or their function, were abnormal. The patients who submitted to the eye treatment had refractive or muscular anomalies, or both, and were treated by glasses, prisms, or tenotomies. His results were as follows: 8 cases had no return of the convulsions or other indications of a continuance of the disease for a period of from two to six years; 6 were benefited in a general way and the attacks rendered less frequent; one relapsed as soon as the glasses were withdrawn; one received no benefit; one, a case aggravated in every respect, could not be held under observation. The same author reports [21] a case of epilepsy cured by correcting an error of refraction, the inherited neurotic taint being especially prominent in the family of the patient. A man, aged twenty-four, complained of mental confusion, flushing of the face and vertigo in attempting to read, and remarked that the symptoms disappeared when the eyes were closed. At times he was dull and morose, and at others brilliant and exemplary. The diagnosis was that of "petit mal with impulses." The patient had been confined in an asylum at intervals for two years on account of epileptic seizures. Examination of the refraction showed hyperopia .50 D. of manifest, 2 D. of latent; esophoria 8 degrees. By restoration of the balance of the ocular muscles by prism exercise and correction of the total H., the man was relieved of his pitiable condition and was made strong and well. The patient's mother suffered from neurasthenia and "masked" epileptic convulsions, vertigo and unsteadiness of gait. She had H. 2 D., HAs. 1 D., and a muscular error. After the optical defects were fully corrected she, also, became well. The patient's sister was emmetropic with neurotic tendencies. A brother had been an inmate of an asylum for some form of insanity. Colburn says, "The hereditary hyperopic error of refraction was responsible for all the symptoms of progressive cerebral disease, up to insanity." C. M. Capps records two cases.[22] One was that of a girl of nine who consulted him one year before reporting the result of the treatment. She had three to six epileptic convulsions every month since four years of age. Since entering school the seizures had become more frequent and violent. The seizures ceased while the accommodation was paralyzed with atropin, and remained absent during the year, while wearing correction for M. and MAs. The second case was a girl of eight who, after correction of H. and HAs., had complete cessation of epileptic attacks. W. F. Connors describes the interesting case of a young man[23] who was cured by wearing correction for high H. Removal of the glasses was immediately followed by the prodromes of an epileptic convulsion. Reber[24] narrates four cases that are worthy of brief description. Case 1, married woman, aged thirty, high hyperopic refraction combined with muscular imbalance, treated by spherocylinders and prisms; result, "without any suggestion that her epilepsy might be benefited and with the withdrawal of all drugs, this woman was rescued from a future that was very gloomy at best. She had been having two or three seizures weekly, when she would fall to the floor unconscious and remain in a stupor for an hour or more. To be brought from that condition to the present one (having one fleeting fit a month, without loss of consciousness) is practically a cure." Case 2, unmarried woman, aged twenty, in apparently perfect health with the exception of nocturnal epilepsy, having had for a long time 12 to 14 seizures every week. The frequency of the attacks was reduced to one per week by wearing myopic cylinders. Later, prisms were added to her glasses. After wearing the combination for three months, she reported that she was virtually free from attacks, having had them only at great intervals and very lightly." Case 3, a strong, active youth of twenty. After reading several hours in the evening he would suffer on the next day several attacks of petit mal. For several years he had the attacks at irregular intervals, sometimes one or two, sometimes several weeks apart. Treatment of muscular imbalance by tenotomies reduced the seizures to one or two slight attacks in over two years, so that, to all intents and purposes, he is a cured man. Case 4, a man of twenty-one, one of a family of eight children. "There are several neurotics in the family, and the general

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[16] The Relation of the Cervical Sympathetic to the Eye, Ophthal. Sec., Amer. Med. Asso., 1903.

[17] Trans. Penna. State Med. Soc., 1902.

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[18] Functional Nervous Diseases, 1887.

[19] Reported in "Eye-Strain in Health and Disease," 1897.

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[20] Chicago Medical Record, 1894.

[21] Peoria Medical Monthly, February, 1888.

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[22] New York Medical Journal, September 16, 1899.

[23] Medical News, November 4, 1893.

[24] Pennsylvania Medical Journal, November, 1902.

ancestral history is studded with neurasthenia, hysteria, and chorea, but no epilepsy or alcoholism. At seventeen, he commenced to have epilepsy, having two seizures within 24 hours of each other every three or four weeks. Under partial prismatic correction of exophoria and withdrawal of drug treatment the attacks ceased for four months, when two very light attacks were induced by excessive reading several nights in succession. Instead of the 8 to 10 attacks he would have had within the 19 months, taking all the time heavy doses of bromides, he had two very light seizures, almost imperceptible, and that without any drug treatment at all." F. G. Murphy writes [25] that in the past two years he had five epileptics under his care, four of whom had defective eyes. Two of these were completely relieved of all their epileptic symptoms by the correction of their optical defects. The remaining two wore their correction with relief of ocular symptoms, but none whatever from epileptic seizures. Ranney reports [26] results of eye treatment in 29 cases: 7 completely cured, 3 practically cured, 9 improved, 3 not improved, 4 not counted. Gould [27] claims to have cured six cases, and in all "the cure was immediate, certain and complete and persisted for several years." He agrees with most ophthalmic surgeons that in exceptional cases the ocular condition may be the pernicious factor that maintains the epileptic manifestations. The obligation is imperative that in every case of epilepsy the condition of the eyes should be carefully considered. In the Spratling and Gould report of the eye examinations and results of eye treatment in 78 selected cases of epilepsy, who were patients in the Craig Colony,[28] the same differences of opinion that characterizes similar reports are found. Marlow [29] corrected the refraction of a neurotic patient. The patient gained 20 pounds in weight as a result of wearing the glasses, and at the end of five years continued free from the intense nervousness and occasional epilepsy that had baffled many physicians for some years. Coover details 15 cases [30] of reflex irritation accompanying eye-strain. Among the prominent symptoms, all of which were relieved by correction of refractive error, were attacks of convulsions resembling epilepsy caused by continued use of the eyes at the near point. Elmer [31] recited instances of patients afflicted with epileptic seizures, of mental conditions approaching insanity, of chorea, and of petit mal, who were entirely relieved by correcting glasses.

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[25] Trans. Iowa State Med. Soc., vol. xvi., 1898.

[26] New York Medical Journal, January 2 and January 9, 1897.

[27] Journal of the American Medical Association, July 5, 1902.

[28] American Medicine, April 9, 1904.

[29] Medical News, July 4, 1897.

[30] Medical News, October 26, 1895.

[31] Amer. Jour. Ophthal. and Otol., April, 1896.

Having thus presented some of the evidence published in advocacy of the treatment of the eyes as a means of cure of epilepsy, the other and negative side claims attention. Peterson[32] does not remember to have seen among several hundred epileptics a single case of genuine reflex epilepsy. Gowers[33] says, "It is extremely rare to meet with any evidence of a reflex cause in the system of the cerebrospinal nerves and in the few recorded cases there has been a manifest and strong predisposition." Starr believes that reflex neuroses of an epileptiform type are exceedingly rare, and Dana says "the importance of reflex irritation has been much overestimated." Sachs does not even mention reflexes among the causes of epilepsy, except in a footnote, where he says the disease is rarely established by peripheral irritation. Concerning eye conditions, he says that they may cause single attacks, but they surely are not the cause of epilepsy. He asserts that Ranney does not cure cases, and makes the statement on the ground that Ranney has not observed them long enough. Both Dana and Sachs reiterate these opinions in their latest published articles.[34] Féré[35] as late as 1897, in his full chapter on epilepsy, says that "errors of refraction, necessitating as they do efforts of accommodation, may cause epilepsy," and, in the section devoted to treatment, "anomalies of refraction must be corrected." He does not include anomalies of the ocular muscles among the causes. Gowers, in his monograph,[36] is silent on the subject. Sinkler and Pearce,[37] in their analysis of 315 cases, also pass ocular causes by without comment. H. A. Hare[38] quotes Stevens as to the relation of muscular anomalies and refraction errors to the production of epilepsy, and says that thus far Stevens's remarkable cases have adduced no evidence in rebuttal, but in the section on treatment, he does not refer to the ocular treatment, as one of the means of relieving epilepsy.

Thus, it will be seen that the proof of the causal relations of affections of the eye to epilepsy rests entirely with the ophthalmologists. The ablest among the neurologists barely refer to the eye as a cause, or omit it altogether. An unbiased opinion formed from a study of the literature on both sides of the question, and expressed tersely, would be that exceptionally the so-called reflex and idiopathic epileptic convulsions may be cured by correction of ocular anomalies.

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[32] New York Medical Journal, July 4, 1896.

[33] Mental Diseases.

[34] Medical News, July 30, 1904.

[35] Twentieth Century Practice, vol. x.

[36] Epilepsy and other Convulsive Diseases, 1901.

[37] Trans. Med. Soc. State of Penna., 1897.

[38] Epilepsy, its Pathology and Treatment, 1890.

Treatment.—Ocular examination conducted with patience, ability, and thoroughness should be made in every case of epilepsy, including all its forms. In a large proportion, the result of ocular remedies, both on the violence and frequency of the seizures, will demonstrate only its negative value; in a smaller proportion, the seizures will be permanently arrested. It would seem to the writer that a distinction between epilepsy, epileptiform convulsions, petit mal and grand mal need not be drawn when we are face to face with the problem, What shall we do with this case of convulsions? The prognosis of the affection is universally admitted to be unfavorable, and why should we decline to investigate and remove all possible sources of peripheral irritation, whether the symptoms are said to belong technically to the so-called "true" epilepsy or are only epileptiform in character? The therapy of drugs has failed to cure. The bromide treatment, that most generally resorted to, is admittedly palliative only. By dulling the cerebral forces, outbreaks or crises of cerebral activity are held in abeyance. The advocates of the bromide treatment recognize its value in relieving symptoms, but do not claim for it curative power. Brain surgery accomplishes a little. Da Costa says that 4 per cent. of all epileptics are cured by operation on the skull. The removal of reflex irritation arising from atrophied eyeballs, nasal disease, old cicatrices, degenerated testes, and other lesions, has been followed by relief of the convulsions, and, if well authenticated reports of cure by eye treatment are to be credited, a knowledge of the ocular conditions will suggest methods of treatment that we cannot afford to neglect.

The examination of the eye includes, first, *refraction,* and, second, *the extraocular muscles.*

1. **Refraction.**—(For detailed method of diagnosis and treatment of refraction errors, the reader is referred to one of the text-books on that subject.) (*a*) *Hyperopia and hyperopic astigmatism.* Under full paralysis of accommodation in individuals less than forty-five years old the total error is determined, and as near a full correction as will be tolerated, ordered, and worn constantly. Thus, the unusual exercise of accommodation, and the undue stimulation of the extraocular muscles dependent upon it, are removed; the congestion of the uveal tract so commonly associated with accommodative asthenopia, reduced; the circulation of blood and lymph restored to the normal and the excessive stimulation of the ciliary nerves, and consequent overaction of the other branches of the third nerve, relieved. In those

cases in which the accommodation conceals a latent error of refraction, suggested by the lack of harmony between the degree of refraction as determined by the ophthalmoscope, or ophthalmometer, or a difference of refraction between the near and far points, full paralysis of the ciliary muscle should be obtained by stronger solutions of the cycloplegic and its continued application. By these means the eye is fully rested, the degree of the true static error determined, and the causes, resident in the ocular tissues or the result of overuse, which contribute to the production of reflex symptoms, eliminated.

(*b*) *Myopia and myopic astigmatism.* The influence of increased refractive power, in causing reflex phenomena, has already been alluded to. By spasm of the accommodation, by the unconscious non-use of the accommodation or the disruption of the relation of accommodation and convergence, local and constitutional symptoms of eye-strain may be produced. Myopia is, however, seldom named as a cause. As a rule myopic patients do not complain of asthenopia or other evidence of eye-strain, but only of indistinct distant vision. There is a gradual increasing tendency to divergence of the visual axes which is to be controlled, and its further development into an actual divergence prevented, by the muscles of convergence. The maintenance of binocular single vision by the ever-diminishing power of convergence, leads either to the reflex neuroses in neurotic patients, or to dissociation of the eyes in the act of vision. The real defect, as in hyperopia, can be detected only through cycloplegia, but unlike hyperopia, the correction selected with mydriasis is apt to be stronger than the actual degree of myopia. Post-mydriatic examination and the ordering of glasses after complete return of the accommodation, in order that its power may be measured and allowance made when advisable, is essential to a practical and useful correction. In all cases, the examination should be repeated, at intervals of one or two years, and changes in the refraction followed by corresponding changes in the glasses.

(*c*) *Presbyopia.* Comparatively few cases of neuroses are to be attributed to the strain of the eyes from failure of the accommodation due to stiffness or loss of elasticity of the lens, or to the failure of contractility of the ciliary muscle. Yet such cases have been reported, and marked amelioration of distressing symptoms has followed the use of carefully selected glasses. The strain arises from the expenditure of nervous energy in the effort to focus the eyes for small objects at 33 cm. distance, and because of the excessive innervation to, and response of, the muscles of convergence without an equivalent response of the ciliary muscles. The strain may induce, in individuals predisposed to nervous affections, the identical phenomena that are witnessed in younger persons who suffer from eye-strain. The correction of presbyopia is a simple matter. Having learned the refraction of the eye, glasses should be given which supply the deficiency of accommodation so that the patient will read without discomfort at 33 cm.

2. **The Extraocular Muscles.**—In all cases of insufficiency of the extraocular muscles, readily determinable by the usual tests, and by the relations which the findings of the tests bear to the relative strengths of adduction and abduction, the true static refraction must be learned and the correction of it worn long enough both to make certain that the symptoms are not due to ametropia and to exert its beneficent influence on the muscular tensions. In persons too young to wear glasses, long continued atropinization may be effective. Such preliminary treatment of muscular anomalies before resorting to prisms or operations is imperative.

(*a*) *Esophoria* is a tendency to cross the visual axes. The relatively weak divergent power is insufficient to maintain binocular single vision for all distances without undue stimulation, and in order to obtain relief, treatment should be directed to the reduction of the demand on divergence. This may be accomplished by (1) the internal exhibition of nerve sedatives and simultaneous exercise of the muscles of abduction by prisms and the stereoscope, or the amblyoscope of Worth; and (2) by prisms, bases out, the degree of the prism to be determined by the amount of esophoria. The prisms may be incorporated into the ametropic correction, or the latter may be decentred so that it has prismatic action, or they may be worn separately. Although this method is occasionally serviceable, it is open to the objection that it can only be useful by allowing the visual axes to *turn* inward—*esotropia*. It is only a crutch for the disabled muscles, and is not instrumental in restoring the muscles to equilibrium. Moreover, the limit of the usefulness of prisms is soon reached. Esophoria develops under their action, either by uncovering a part of the defect hitherto latent, or by actually creating a greater defect. Notwithstanding this theoretic objection, cases that resist cure by complete and continued rest of accommodation, sedatives and abduction exercise, should wear prisms before resorting to the final means of cure. (3) Operation. Shall tenotomy or advancement be chosen? Esophoria, dependent upon hyperopic refraction, is not primarily induced by weakness of divergence, but by excess of convergence, a physiologic sequence of the inordinate action of the accommodation. Hence tenotomy of one or both of the interni is the logical procedure. The operation should be performed with great care and frequently interrupted to note, by testing with the Maddox rod or other means, the effect of work already performed. Free division of the tendon and adjacent structures is unjustifiable. Partial or graduated tenotomy only, as recommended by Stevens and Ranney, is the proper operation. These surgeons state that equilibrium can seldom be secured after a single operation, and claim that, should more defect be uncovered as time goes on, a second, third, or more graduated tenotomies should be performed, the necessity for the subsequent operations being indicated by a return of the symptoms.

(*b*) *Exophoria* is a tendency of the visual lines outward. The correction of ametropia, the use of nerve stimulants in large doses, prism exercise of the weakened convergence, the restriction of the time devoted to the near use of the eyes, and other hygienic measures referred to under esophoria, will, in the majority of cases of exophoria, obviate the necessity for operation. Should, however, the symptoms resist this treatment and the wearing of prisms, advancement or resection of one or both interni should be preferred to tenotomy of the externi. Thus, deficient adduction may be strengthened and made to compensate for the relatively stronger abduction.

(*c*) *Hyperphoria* is a tendency of the visual axes to separate vertically, or of one visual axis to assume a plane higher than that of its fellow. This is the most frequent of the anomalies of the muscles of the eye to cause reflex neuroses, and to Stevens is due the credit of scientifically and practically elaborating the causal relations, and of bringing the subject impressively to the notice of the profession. As in the other muscular anomalies, this is dependent in the large majority of cases upon errors of refraction. The principles of treatment are essentially those of other defects of co-ordination.

CHOREA.

Involuntary tonic or clonic contraction of a muscle, or of groups of muscles, is a symptom of an organic disturbance of the cerebrospinal system, or a reflex perversion of function of one or more of the organs of the body. The former, or "true" chorea, is, according to some authorities, an acute infectious disorder, variable in its limitations, nature and course, according to the character of the basic disease. The latter "habit," reflex or symptomatic chorea, is a purely functional affection, the development of a peripheral irritation.

The word chorea has been interchangeably used by writers in describing true chorea and habit chorea, affections that are essentially different in their etiology, prognosis, and treatment. In consequence of this vagueness of definition, much confusion has arisen as to the true significance of chorea, and unprofitable controversies, more or less bitter, have been waged in medical journals.

The relation of eye-strain to the causation of true chorea is practically denied by authors of wide experience—men whose opinions are worthy of the greatest consideration. Thus, Lees,[39] in an accurate and presumably full description of the pathology and treatment of this disease, makes no mention of eye-strain and this possible cause is not alluded to by the speakers who discussed his paper. M. Allen Starr scientifically studied 1400 cases treated in the Vanderbilt clinic, and Krafft-Ebing 200 cases,[40] without finding a single subject in which the cause could be attributed to eye-strain. Aldrich,[41] in an impartial study of the subject, criticises Stevens's and Ranney's reported cases of the cure of chorea by eye treatment, by stating that their cases were habit spasm, or possibly hysterical chorea, and not one case was true chorea. "It was merely a question of mistaken diagnosis." Baker's report of his cases[42] was also criticised by Aldrich[43] in these words: "He did not believe that the winking and grimacing of children as described by Baker to have been relieved by glasses was chorea, and that chorea proper could not be caused by eye-strain since it was undoubtedly an acute specific nervous affection." Starr further says "while it is possible that local twitching of the muscles of the eyes or face or neck may be produced by eye-strain or irritation of the nasopharynx, true chorea is never, in my opinion, produced by these causes, and treatment directed to the relief of the anomalies of the eye muscles is useless."

The chorea of eye-strain is essentially that described years ago by S. Weir Mitchell as "habit chorea," and more recently by Osler[44] as "simple tic" or habit spasm. According to the latter authority, it is to be carefully differentiated from chorea with superadded psychical phenomena, and from complex and co-ordinated chorea. Further, as Wilbrand and Saenger have pointed out,[45] habit chorea should not be confounded with or mistaken for the reflex chorea, or choreic movements, of the orbicularis palpebrarum (blepharospasm), of the frontalic and the corrugator supercilii, which are dependent upon irritative or-of a needed glass or the correction of a muscular error will soon decide the etiology. A large proportion of choreic patients have hyperopia and astigmatism, but 95 per cent. of all individuals have an error of refraction; therefore, it would appear that frequency of ametropia in choreics has little or no bearing on the relation of cause and effect. Cheney[49] examined 23 cases of chorea, and found emmetropia 2, hyperopia 15, hyperopic astigmatism 3, myopia 1, myopic astigmatism 1, unknown 1, esophoria 8. His conclusion was that "patients frequently recover while undergoing no treatment whatever, but the marked improvement or rapid recovery following the correction of refractive or muscular errors in some of these cases makes it seem more than probable that the ocular defects were the exciting cause of the disease." The same writer reports the case of a boy, eight years of age, who, since his fifth year, had never been free from chorea, although the twitchings were slight at all times. The patient complained of headaches for a number of years. Two months previous to Dr. Cheney's examination it was noticed that the left eye turned inward; incidentally the twitchings became considerably aggravated, the movements involving both the upper and lower extremities, neck, and face. An examination of the eyes under atropia revealed, in addition to the convergent strabismus, hyperopia of 1.50 D. in both eyes. A cure was effected by correcting the refractive error and tenotomizing the internal rectus. De Schweinitz examined 50 cases of chorea in the Orthopedic Hospital and Infirmary for Nervous Diseases, and in 77 per cent. of these patients hyperopia and hyperopic astigmatism were the predominating condition of the refraction, but "evidence seems lacking that the refraction error is the basal cause of the chorea." Of 118 cases examined by Stevens in his private practice, all but 5 had defective refraction, and a considerable number muscular insufficiencies.[50]

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[39] British Medical Journal, 1903, 11.

[40] Festschrift in honor of A. Jacobi.

[41] Arch. Pediat., 1903, xx.

[42] American Medicine, March 14, 1903.

[43] American Medicine, May 9, 1903.

[44] Arch. Ped., January, 1897.

[45] Die Neurologie des Auges.

Roosa[51] says that of 118 cases of chorea, 78 had hyperopia, 13 had hyperopic astigmatism, 5 had mixed astigmatism, 6 were myopic, and 11 had myopic astigmatism.

These figures, and others of like import, simply show that refraction errors are extremely common among choreics, but they do not prove that ametropia is the primary causative factor in producing chorea. It is, however, reasonable to suppose that accommodative or extrinsic muscular strain, like irritation in any other part of the body, may aggravate the symptoms of chorea or any other nervous affection. Eye-strain is a waste of nerve force and escape of energy, a consumption of power so easily prevented, so amenable to treatment, that whenever even a suspicion of its presence exists an examination should be demanded.

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[49] Boston Medical and Surgical Journal, February 20, 1890.

[50] The reader is referred to the remarkable array of case histories in his book on Functional Nervous Diseases.

[51] Transactions of the New York Academy of Medicine.

VERTIGO.

Vertigo, subjective or objective, is a symptom of many diseases, organic and of known pathology, and of functional affections which have no recognizable or defined lesions. It is found, for instance, in disease of the inner ear, irritative intracranial affections, disease of the heart and blood-vessels, of the blood (toxæmia), and other affections by which equilibration is temporarily or permanently destroyed.

Vertigo may be reflex. Mills says, "Irritation may be reflected to the apparatus of equilibration from almost any part of the body, near or far. Reflex vertigos are termed ocular, aural, nasal, pharangeal, gastric, gastro-intestinal hepatic, uterine, or ovarian, according to the organ which seems to be the fountain-head, but if they are truly reflex disorders the mechanism of their production is practically the same in all cases,—namely, a disturbance of equilibrium produced by the reflection of an abnormal stimulus upon a more or less non-resisting equilibratory apparatus." It will be seen that the presence of vertigo or dizziness is of little value in determining the nature of the underlying affection. The diagnosis is, necessarily, often one of exclusion. The appearance of vertigo incidently with the performance of the function of one or more organs of the body will, however, often indicate its probable origin.

Ocular vertigo is the result of causes identical with those that are active in giving rise to other symptoms of irritation or exhaustion of the nervous system,—namely, accommodative strain and muscular insufficiencies. Any uncorrected error of refraction, or presbyopia, or heterophoria in an individual predisposed to nervous affections may be responsible. Vertigo occurs in about 12 per cent. of all cases of asthenopia, including both accommodative and muscular,[52] in which no disease of the visual apparatus can be shown. It is common also in those that have asthenopia from wearing inaccurately adapted lenses, either in the strength of the lens or in the adjustment of the axis of the cylinder.

That vertigo may be a symptom of eye-strain was shown by Thomson and later by the joint papers of Thomson and Weir Mitchell. Their statements have been verified in thousands of cases, and are being daily shown to be true in the routine work of the oculist.

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[52] Marlow, New York Medical Journal, July 13, 1897, gives 29 per cent.

Ocular vertigo is, with few exceptions, associated with, or consequent upon, the use of the eyes for near work. It is purely subjective, the patient becoming dizzy and feeling as though he would fall. Objective vertigo, or the apparent movement into unreal or unnatural positions and relations of stationary objects, is rare. Ocular vertigo is momentary, disappearing with the cessation of the use of the eyes and recurring with resumption of work. The relation of its oncoming to the performance of close work depends, as in other forms of asthenopia, upon the relative strength of the accommodation and of the extrinsic muscles, as compared to the degree of the ocular defect. The condition of the general health has also much to do with the advent and frequency of the attacks of dizziness. The attacks recur with greater frequency in those who are weakened from disease or overwork, who lead sedentary lives, who are confined many consecutive hours to their desk or sewing-machine, or who have some other affection than asthenopia. And conversely, the attacks are less severe and frequent in the healthy and well-fed and among those who observe the rules of hygiene.

Dizziness seldom exists alone as a symptom of eye-strain, but is associated with local asthenopia and other reflexes, particularly nausea. The gastric disturbance may be secondary to the vertigo or primarily induced by the cause of the vertigo. The combination of dizziness, nausea, and vomiting is not accidental, but an intimation of the association of eye-strain with functional stomach disorder. All these symptoms may be, of course, the result of eye-strain alone, particularly when they occur with, or after, use of the eyes.

Immediately preceding and during the attacks of vertigo, the condition of the cerebral circulation is doubtful. It is probably not the same in all sufferers, and not always the same in one individual. Whether it be one of anæmia or hyperæmia, the circulatory disturbance is momentary and probably limited to an extremely small central area. Upon removal of the cause or cessation of the use of the eyes, the dizziness is allayed, because the cerebral disturbance, whatever it may be, is removed. The effect of repeated irritation of the centre of equilibration is deleterious, and while for a time the affection may be functional, constant repetition must lead to organic change with permanent loss of normal function and deterioration of the mental faculties.

Diagnosis.—The characteristic features of ocular vertigo are its subjectiveness, transitoriness, and association with the act of vision. In order to test its ocular origin, the patient should abstain from all sources of eye-strain, either by voluntarily giving up the use of the eyes or by induced paralysis of accommodation. Should the vertigo persist, the cause must be looked for elsewhere than in the visual apparatus. The subsidence of the vertigo, on the other hand, indicates that it is of ocular origin. Vertigo, dependent upon errors of refraction and functional anomalies of the muscles, is, strictly speaking, ocular vertigo, and should not be confounded with the vertigo of organic disease of the ocular nerves, in which the lesion is orbital or of the cerebrospinal system. For example, hemianopsia, limited fields of vision, diplopia, the apparent distortion of objects by imperfect retinal images, hallucination of vision, etc., frequently are associated with vertigo. Vertigo of brain tumor, in which the optic or other ocular nerves are involved, is also not to be classified as ocular vertigo.

Treatment.—Correction of errors of refraction and the restoration to balance of the extrinsic muscles are imperative. Rest of the eyes by abstinence from the usual occupations, tonics, and other general measures, will give temporary relief, but permanent cure can be established only *by removal of the defect of the visual apparatus which is the cause of the eye-strain and vertigo.*

A few illustrative examples from the journals and from my own histories are appended.

Colburn[53] quotes the case of a farmer who suffered from severe headaches, vertigo, and nausea. He was examined under atropia, and was found to have a hypermetropic astigmatism in each eye, besides a hyperphoria of one degree and a manifest esophoria of eight degrees in the right eye. He was given his correction and advised to return if the symptoms did not abate. Four and a half years later he appeared, not having complained of any discomfort until recently. No change was found necessary in his glasses. The hyperphoria had increased, however, to four and a half degrees, but the esophoria remained the same. The right superior rectus was tenotomized and the inferior of the same eye advanced, the result being that a hyperphoria of one degree remained and the esophoria almost entirely disappeared. He was seen two months later, when he stated that the symptoms from which he suffered had not returned.

The same author[54] gives details of four classes of intractable asthenopic headaches, with insomnia, vertigo, indigestion, and, in one case, petit mal, persisting in spite of careful refraction. Correction of muscular anomalies gave relief in four cases.

Fulton (in *Northwestern Lancet,* 1900) reports the case of a business man who suffered from almost constant vertigo. For two years he was treated at sanitariums in America and Europe without obtaining relief. Examination showed a heterophoria. A tenotomy of the inferior rectus was done, with the result that all symptoms of vertigo completely disappeared, and, up to one year after the operation, had not returned.

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[53] Clinical Lectures on Diseases of the Eye.

[54] Clinical Review, May, 1901.

Carter[55] reports the case of a young man, the son of a wealthy banker, who was obliged to relinquish his studies at Oxford because of his inability to read without incurring diplopia, followed by vertigo, and, if continued efforts were made to read, by sickness, palpitation of the heart, and intense headache. Both eyes were found to be myopic, but otherwise the media and fundi were normal. He was refracted and suitable glasses adjusted, with the result that all symptoms from which he had suffered disappeared almost immediately and had not returned when seen about three months later.

Case 1. Mrs. T., aged 29 years. Complained of attacks of vertigo, lasting thirty minutes, followed by severe headache and occasionally by vomiting. The dizziness commenced at 15 years of age, and recurred every week or twice a week for the past 12 years. The attacks were attended with numbness of the fingers. V=6/6. R. hyperphoria ½°, esophoria ½°. She was relieved by full correction of HAs.

Case 2. Mrs. T., 48 years, momentary dizziness while working as a weaver or while walking. The trouble commenced simultaneously with the wearing of glasses for the correction of compound HAs., and presbyopia. Cure was effected by addition of the prismatic correction of 2° of hyperphoria.

Case 3. Mrs. K., 46, nearly constant headache; efforts to see near objects invariably induced vertigo, nausea, and vomiting. Correction of high-grade mixed astigmatism and presbyopia gave entire relief from her distressing symptoms.

Case 4. Mrs. L., 27. Could not read half an hour without headache and dizziness. She had always considered herself nervous and excitable. Headache and vertigo were cured by 25 D. less than full correction, which under cyclopegia was found to be

R. + .75 ⊃ + .25 ax. 15°
L. + .50 ⊃ + 1. ax. 165°

Case 5. Mary F., 11. Neuralgia in left side of face and dizziness, aggravated by near use of the eyes. Print seemed to grow small and recede. She also suffered from constipation, pain in back of neck and across the shoulders. These symptoms were cured by:

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[55] Transactions of the Clinical Society of London, vol. viii.

R. + 2.25 ax. 90°
L. + .50 ○ + 2.25 ax. 90°

Multiplication of cases is unnecessary. It is universally admitted that vertigo may be a symptom of ocular strain, and it is cured by lenses to correct refraction and presbyopia, by prisms, and by operations designed to overcome abnormal tendencies of the ocular muscles. It is, however, interesting to note that practically identical ocular errors give rise to no reflexes in some patients, while in others they may cause vertigo, gastric disturbances, or a complexity of symptoms, which apparently have no connection with the use of the eyes. For example, May F., a sister of Case 5, had much the same error with entirely different symptoms. Her refraction was R. + 150 ○ + 1.50 ax. 90°; L. + 1 ○ + 3 ax. 105°. She complained only of occasional headache and confusion of print in reading. Edward F., a brother, made no complaint at all except imperfect vision, and his defect was

R. —.50 ○ + 450 ax. 100°
L. —.50 ○ + 1 ax. 90°

To revert to a principle, already mentioned, the optical or muscular defect does not decide the character of the reflexes.

GASTRIC DISTURBANCES.

The gastric symptoms of ocular strain are those of so-called functional stomach affections,—namely, flatulency, delayed digestion, hyperacidity, nausea and vomiting, and the natural sequence of indigestion — constipation. They manifest themselves in different intensity, according to the kind and degree of optical defect and according to the individual disposition. They may develop in persons whose digestive organs would be, under normal ocular conditions, in perfect functional activity, or in those whose digestive tract is disordered, as a result of other cause than ocular. They also occur as the result of disease of the digestive tract, and are aggravated by the use of the eyes. The history of their appearance, severity, continuance, and recovery corresponds in every particular to the history of other forms of asthenopic and reflex neuroses, appearing simultaneously with the commencement of close (or far) application of the eyes, and in a few minutes increase in intensity until the occupation is discontinued and the patients' eyes are put at rest. The symptoms persist during the period of eye-strain, and recover only after abstinence from work and possibly seclusion in a dark room.

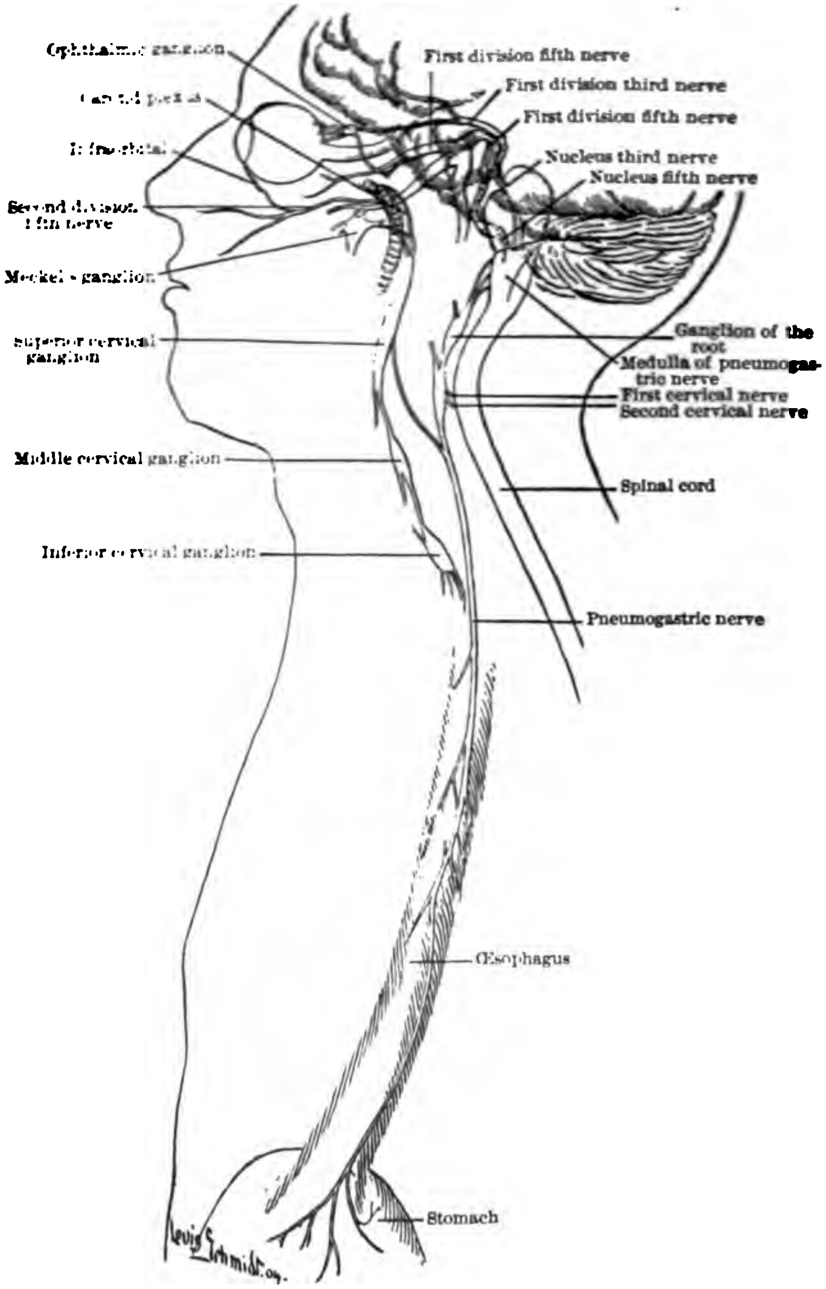

FIG. 1.—A schematic diagram of the cerebrospinal and sympathetic nerve connections between the eye and the stomach.

The schematic anatomical explanation upon the opposite page (Fig. 1) of the origin of the gastric disturbances from irritation of the fifth pair of nerves helps to elucidate their ocular causes. It is, indeed, applicable to lesions occurring anywhere in the course of the fifth nerve.

That indigestion may be due to eye-strain there can be no reasonable doubt. Too many authentic cases are on record to permit of scepticism. Unquestionably, hasty eating, imperfect mastication, ice-water, resumption of mental or physical work immediately after partaking of heavy meals, indifference to the laws of hygiene, and, in a word, too little thought of health, are contributory causes. Nerve energy that should be spent in stimulating the different organs of the body to perform their functions is diverted to the visual apparatus. Focussing rays of light from near or distant objects and the adjustment of the musculature of both eyes for single binocular vision for more or less prolonged periods demand an excess of nerve force that cannot be spared without sacrificing the functions of other organs. The causative relation of eye-strain is seldom suspected. Treatment directed to the abdominal organs gives temporary benefit only. Permanent recovery ensues only after correction of all optical defect.

The ocular factor in the etiology of gastric disorder has been insisted upon by oculists for many years.

The following histories are not uncommon: In the article by Thomson,[56] already alluded to, nausea is included in the description of asthenopia induced by astigmatism.

M. X., lawyer, aged 30, complained of occasional blurring of distant vision, photophobia, chronic conjunctivitis, and headache, all of which symptoms he attributes to indigestion. Under mydriasis the refraction was $+2 \bigcirc +1^{\circ}$ ax. 90°. Correction of this defect cured the local asthenopia, headache, and indigestion. The patient had submitted to medicinal treatment for indigestion for years, and was not a little surprised at the cure by glasses. Miss M. complains that she cannot use her eyes day or night without pain. Is nervous, irritable, and dyspeptic. After correction of HAs., muscular exercise by prisms, nerve tonics, and, finally, tenotomy of one of the superior recti muscles, she reported that she could read or sew several hours comfortably and felt herself " a new creature."

Gould [57] gives the history of a case of flatulent dyspepsia of twenty years' standing cured by the application of proper lenses. The patient complained of certain ocular and cerebral troubles, asthenopia, difficulty in near work and the like, besides the gastric disorder, though it was the latter which gave her the greatest cause for worry. Medicines never gave her any relief. There was absolutely no hysterical element in the case. The reflection of the ophthalmoscopic mirror into the eye caused such an instantaneous and frightful paroxysm of eructation that, for fear that violent emesis was about to take place, the examination had to be deferred. It was highly interesting to note that the application of suitable lenses in a trial frame as effectually quieted the gastric volcano as the ophthalmoscopic mirror had caused it. Glasses were prescribed, with the result that the patient improved each day. She stated that she could not leave the glasses off a minute without gastric symptoms. That the condition was purely a reflex neurosis was clearly evident, because removal of the glasses from before her eyes while fixing an object caused the eructations to reappear like a flash, and again disappear when the glasses were put in place. Knapp says, in the treatment of so-called gastric troubles, flatulent and other dyspepsias, indigestion, etc., the neutralization of errors of refraction and restoration of normal muscle balance yield such good results that ophthalmogists are justified in constantly advising ocular treatment. Ayres[58] reports a case of a healthy, well-developed boy of 15 years, who complained of nausea when in school, in church, or in any assembly of persons. The sensation became so marked in school that he was obliged to discontinue his studies. Ophthalmoscopic examinations revealed a hyperopia of 1.5 D. in each eye. He was refracted and ordered a +1 D. to be worn constantly. The results were immediately manifest. The nausea was at once relieved. Parker[59] says that while, as a general rule, the symptoms produced by refractive errors and ocular imbalance are varied, the headaches are characteristic. The migraine produced by refractive errors begins as frontal, extends to the top of the head, and finally settles in the back of the neck. He quotes several interesting cases:

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[56] Medical News and Library.

[57] Medical and Surgical Reporter, February, 1889.

1. A man, aged 35, complained of being nauseated and of having severe headaches for two or three days each time he audited his books, which was every month. So severe were these attacks and so directly referable to his near work that he was obliged to refrain from excessive use of his eyes for the balance of the month. He had a low myopia for which he used his correction constantly. Muscle test showed one degree of exophoria. By orthopic exercises the divergence disappeared,

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[58] Cincinnati Lancet Clinic.

[59] Physician and Surgeon, May, 1902.

as did the symptoms, after six weeks of training. This patient has been under observation for seven years, and in that time he has been entirely free from his old attacks.

2. A girl, aged 10, suffered daily from intermittent headaches, nausea, and vertigo for six years. Examination showed an exophoria of 9°; although glasses were given to correct the astigmatism present, and adduction was developed by exercise, the symptoms continued. A complete tenotomy of the external rectus reduced the muscle error to naught. It later went back two degrees, but no symptoms developed and she has had perfect comfort ever since the day of operation.

GENERAL ASTHENOPIA.

Asthenopia, painful use of the eyes, is common to all forms of refractive defects and muscular anomalies, and is, exceptionally, present in emmetropia and muscular equilibrium. One or the other of these conditions is necessary for its development, but, as a rule, both factors are coexistent, and in most asthenopics,—namely, a defect in the visual apparatus and an impressionable and responsive nervous system. The use of emmetropic eyes under unhygienic conditions of light, improper physical posture, unusual or unnatural direction of fixation in healthy individuals, will cause asthenopia, which assumes an exaggerated expression in those who are infirm or predisposed to muscular weakness.

Asthenopia is accommodative or muscular. (It may be both accommodative and muscular.) In the former case the power of accommodation is unable to cope with the disadvantages of ametropic refraction; in the latter the nerve stimulation to the extra-ocular muscles or the muscles themselves are defective. Both forms give rise to local and general symptoms which may be logically referred to ocular causes or which may be so independent of the use of the eyes that their ocular origin is only suspected after exclusion of other possible causes.

The Acuity of Vision in Accommodative Asthenopia.—Lowered vision, or the inability to perceive and name letters or objects that are small enough to be included in the 5′ visual angle, in any distance between the near-point and the so-called infinity (6m.), is not always, and indeed in the majority of cases, associated with or a symptom of asthenopia. Its presence is, however, indicative that the visual apparatus is imperfectly functionating. The defect may lie in opacity of the media, in disease of the optic nerve, from its deep origin in the brain to its expansion at the fundus of the eye, or in an error of refraction. Eliminating disease, as irrelevant to the subject of this chapter, we are limited to a consideration of Hyperopia, Myopia, and Astigmatism.

(*a*) *Hyperopia* (H).—The acuity of vision depends upon the degree of H., the power of accommodation, and the distance from the eye at which the test is made. Young persons in good health may conceal, by ciliary contraction, 2 to 3 D. of hyperopia for all distances. 3 D. may be consistent with full acuity for far, but not for near. Higher hyperopia than 3 D. in the great majority of persons, even the very young, means lowered vision, and is therefore one of the symptoms of an optical defect. In advancing years the contractility of the ciliary muscle becomes reduced and the elasticity of the lens lessened. Latent H. becomes manifest H., and the acuity of vision appreciably declines. Between forty and forty-five years of age in Em. distant V. may remain full, but the near-point has receded, and full acuity for the ordinary reading distance is no longer present, or, if present, is maintained for a few minutes only. In H. the distant V. always suffers with the receding of the near-point. In myopic refraction, distant and near vision are practically unchanged by failure of accommodation.

(*b*) *Myopia* (M).– It will be seen from these statements that the acuity of vision depends in part upon the distances at which the tests are made, and that no examination is complete unless the determination of the position of the near-point is included. Lowered acuity for near is suggestive of hyperopic refraction, although myopic astigmatism may also be a cause. In M. vision is always less than 6/6. The ability to see small objects, such as test types, at the distance at which they are seen by emmetropic eyes, the accommodation being abolished either pathologically or therapeutically, is not incompatible with myopia of low degree. Myopes, however, usually read at a distance nearer than the degree of M. would warrant, since the convergence necessary, in the associated function of both eyes, induces contraction of the ciliary muscles, and this, in turn, increases temporarily the M. Relaxation of the accommodation may not be complete even for 6 M., particularly in individuals accustomed to long-continued near work. Hence M. will be apparently greater than the degree shown under complete cycloplegia. I have seen a remarkable illustration of this fact. A woman, aged 35, had binocular V = 6/60 corrected to 6/6 by —3 D. When she fixed with one eye and allowed the other to diverge, V. improved to 6/6 without a glass.

(*c*) *Astigmatism* (As).—Lowered visual acuity is found in all grades of MAs. and HAs. of 1 D. or over. In spasm of accommodation, by which HAs. is apparently transformed into MAs., the acuity of vision is below the normal and is improved by a minus cylinder before cycloplegia, a plus cylinder during cycloplegia. In mixed As. the myopic meridian may be made more myopic and the hyperopic meridian less hyperopic by contraction of the muscle of accommodation. In either case the acuity of vision is reduced.

Full acuity of vision in the distance is present in most cases of accommodative asthenopia. Therefore, the test so often made, to determine whether the eyes are at fault by test cards as 6 M., is misleading, and the exclusion of the eyes as a causative factor because $V = 6/6$ is incorrect. Should, however, the combination exist of full visual acuity and muscular balance for all distances, it is probable that Em. is present and the symptoms of which the patient may complain are not of ocular origin.

The visual acuity, therefore, is no criterion of the presence of accommodative asthenopia, since asthenopia may be a symptom either in normal or defective vision. It is claimed by some writers, however, that asthenopia is more frequently present in the former than in the latter, and consequently is to be attributed to the constant effort of accommodation necessary to overcome the slighter degrees of ametropia by which vision is maintained at the normal standard. Yet it should be borne in mind that one additional factor enters into the eye-strain in individuals who have eyes so defective that the accommodation is powerless to entirely overcome the defect. The contraction of the ciliary muscle continues as persistently in the higher as in the lower defects, and the imperfect vision is only partly or not at all restored. The mental effect of the unclear retinal images should not be disregarded. The mind is erroneously informed of the actual size, form, and relation of objects, and is led to form wrong conclusions, the effects of which are far-reaching. The discovery of this unconsciously misguided attitude towards the world exerts a baneful influence upon the entire system, tends to melancholy and depression of spirits, social exclusion, and helps towards the development of a misanthrope.

Vision in Muscular Asthenopia.—The effect on vision of the degrees of muscular defect is analogous to that of the degree of ametropia. Vision may be single (binocular) for all distances and at all times; it may be single ordinarily and blurred occasionally, when the blurring may be taken as evidence that the weaker set of muscles are momentarily incapable of maintaining the tension necessary to resist the opposing muscles. Simultaneous with the deviation of the visual lines, the object looked at becomes indistinct in outline or appears double. Vision may

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physical or mental condition which tends to lessen the physical
owers of resistance or to the induction of irritability or undue impressionability of the nervous system. Among the former causes are convalescence from illness, toxæmia, gout, rheumatism, exhaustive hemorrhages (as in childbirth), anæmia, diseases of the blood, sedentary life, and impure air. Among the latter are functional and organic derangements of the cerebrospinal system, resulting from disappointed ambition and desires, anxieties, and a want of proper relation between financial income and outgo. The ocular defect in constitutional asthenopia may be small or entirely absent. The symptoms depend less on the state of refraction or of the muscles, and more upon a deficient muscular and nerve force. The ciliary and extraocular muscles, like other muscles of the body, are unable to respond normally to the demand made upon them, and the nerve stimulation to the muscles, originating in the consciousness, is weaker than the will which governs it. The so-called "retinal" asthenopia may be regarded as another name for constitutional asthenopia. The limit of endurance of the retina to the impression of light rays and that of the optic nerve to conveying the retinal impulse to the cortical centres is reached in a comparatively

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[61] Transactions of the American Ophthalmological Society, vol. vii.

be permanently double, indicating insuperable deviation from each other of the visual lines. Asthenopia of great severity usually accompanies the first and second or latent forms of insufficiency and disturbed mental equilibrium, from the presence of the two images, is the common result of the third form.

The Facial Expression in Asthenopia.—Stevens and others have called attention to the modification of the facial expression, induced by the conscious or unconscious participation of some of the facial muscles in the accommodative and muscular effort to secure clear and single images. The facial expression is determined in part by the ocular defect. For example, the myope by contraction of the orbicularis lessens the vertical diameter of the commissure, and by cutting off the light rays that enter the periphery of the pupil, the circles of diffusion that fall upon the retina are reduced in size. The contracting muscle also modifies the curvature of the cornea by pressure upon the vertical diameter. The same statement holds for myopic astigmatic when the axis of the astigmatism is horizontal, or a few degrees removed from the horizontal. Should the axis be vertical, or nearly so, better vision is obtained by turning the head on its vertical axis. In heterophoria the muscles of the brow are contracted, lining the forehead with vertical or horizontal furrows. In heterotropia the orbicularis of one eye is maintained in a state of constant contraction in order to exclude that eye from the act of vision, or the head is rotated in its vertical or horizontal axis in the direction of the weaker set of muscles. Thus, in paralysis of the third nerve of the left eye, the eye is diverged, and the false image can be merged into the true by turning the face towards the right.

"Wrinkles," or "crows' feet," at the external angles of the commissures are in many persons an indication of eye-strain. In exaggerated cases of ametropia and muscle imbalance, the facial muscular contractions assume the proportion of deformities, and are suggestive of the expression of horror, fright, astonishment, or other emotion. These lines may become permanent, and are often misleading indications of character and disposition.

Causes.—Errors of refraction and anomalies of the extraocular muscles have been alluded to. They are by far the most frequent causes of asthenopia [60] and affect a large proportion of the population of educated communities. The causes are most active between the tenth and fortieth years of life, although no time of life is exempt. Hale[61] says that muscular asthenopia usually has its origin in the brain, depending upon a disturbed innervation or possibly, in some cases, upon a weakness of the power of fusion. In a small percentage of cases asthenopia depends upon either the absolute or relative weakness of the adductors or obliques, or upon the position of their insertions into the sclera. His own observations lead to the belief that want of converging power is the chief cause of this form of asthenopia. Optical defects are of such vast importance to health and intellectual growth that it is incumbent upon every parent or guardian of youth to seek an examination of the eyes of those under their care.

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[60] For correction of these defects and the treatment of accommodative and ocular asthenopia the reader is referred to special works on ophthalmology.

Reflex asthenopia is sufficiently common to deserve more attention than is accorded to it. Painful use of the eyes may be due to nasal obstruction, to dental caries, to throat and nose affections, and to the reflected irritation from disturbed function of the organs of the chest and abdomen. A familiar example is asthenopia from disease of the cavities accessory to the orbit, such as ethmoiditis, rhinitis, and frontal sinus affections, by which the circulation of the orbit and eye is obstructed or the passage of the tears through the nasal duct prevented.

Constitutional Asthenopia.—Painful use of the eyes may result from any physical or mental condition which tends to lessen the physical powers of resistance or to the induction of irritability or undue impressionability of the nervous system. Among the former causes are convalescence from illness, toxæmia, gout, rheumatism, exhaustive hemorrhages (as in childbirth), anæmia, diseases of the blood, sedentary life, and impure air. Among the latter are functional and organic derangements of the cerebrospinal system, resulting from disappointed ambition and desires, anxieties, and a want of proper relation between financial income and outgo. The ocular defect in constitutional asthenopia may be small or entirely absent. The symptoms depend less on the state of refraction or of the muscles, and more upon a deficient muscular and nerve force. The ciliary and extraocular muscles, like other muscles of the body, are unable to respond normally to the demand made upon them, and the nerve stimulation to the muscles, originating in the consciousness, is weaker than the will which governs it. The so-called " retinal" asthenopia may be regarded as another name for constitutional asthenopia. The limit of endurance of the retina to the impression of light rays and that of the optic nerve to conveying the retinal impulse to the cortical centres is reached in a comparatively short time. The retina and nerve become easily fatigued and soon exhausted, giving rise to confusion of images and impaired mental conceptions. The exhaustion is a symptom of the combination of optical errors and of constitutional weakness, and is common in nervous affections, independent of ocular error, such as neurasthenia and hysteria. "The subjects break down easily and are unable to continue work, and have but little initiative." (Dand.)

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[61] Transactions of the American Ophthalmological Society, vol. vii.

Symptoms.—Etymologically, asthenopia means weak sight. Ophthalmologically, the word has come to signify painful sight. The symptoms are local and reflex. Of the former, pain constitutes the principal phenomenon. It is felt in the eye, in the orbit, through the radiations of the first and second branches of the fifth nerve, and the first and second cervical. It follows immediately, or not for some minutes or hours after using the eyes, either for distance or in reading. It is slight or severe, sharp, neuralgic, or dull and boring, intermittent or constant, acute or chronic. It may have, apparently, no connection whatever with the use of the eyes or be originated or increased by reading or other near work. Occasionally ciliary tenderness is present, but in most cases the pain is temporarily lessened or wholly relieved by external pressure upon the eye-balls. Blurring of letters in reading or the needle in sewing, or confusion of points and lines in any near work is due to transiently relaxed accommodation to momentary suspension of convergence or to exhaustion of retinal energy. The time of the oncoming of asthenopia after commencement of the near use of the eyes is, like other forms of asthenopia, dependent upon the power of accommodation, the relative strengths of the extrinsic muscles, and the tone of the nervous system. Hyperæmia of the conjunctiva and lachrymation may be associated with pain or occur independently when the eyes are subjected to strain in the effort for the accurate definition of images, particularly if long continued.

Reflex Symptoms.—Headache is one of the most constant and aggravated symptoms of ocular strain in individuals under 40 years of age. It is a reflex pain of muscular effort and arises from excessive or prolonged contraction of the ciliary muscle, from a disturbance of the normal relation of accommodation to convergence, or from imbalance of the external ocular muscles, intrinsic in the muscles themselves, or in their innervation and possibly independent of the refraction of the eye.

Location.—Some authors[62] assign definite portions of the head as the seat of ocular headache—frontal or temporal, for instance—in contradistinction to the location of headache from other causes. Wood has stated the order of frequency to be supraorbital, deep orbital, fronto-occipital, and temporal.

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[62] Marlow, C. A. Wood.

The exceptions to definite localization are so numerous that but little diagnostic value attaches to the location designated by the patient. In one hundred successive cases of accommodative or muscular strain under my care sixty-eight patients complained of headache, classified under the descriptive term "general," in twenty cases the pain was frontal, two temporal, and in ten occipital. The disproportionately large number of general is partly the result of inaccuracy of localization, observation, and expression on the part of the patient, and to the dissemination of the pain throughout the head, from its origin within the orbit. The distribution of pain must be along the sensitive nerves of the head, which in their course or termination are subjected to irritation. Hence the pain felt outside of the orbit is reflex. Orbital pain, that arising from muscular tension or irritation of orbital nerves, might readily be referred to the temporal and frontal regions of the head through the distribution of the fifth pair of nerves. The exhausted ciliary muscle, through sympathetic fibres or directly, causes an irregular discharge in the region of the third nerve nucleus or an irritation of the necleus itself. Occipital pain, often a symptom of muscular anomaly and of uncorrected astigmatism, is referred, not to the scalp, but to underlying tissues, and is possibly explained by the presence of localized congestion of the vessels and perivascular tissues at the base of the brain, induced by the effort to maintain balance of the extra-ocular muscles or by irritation of the third nerve communicated to the sensitive nerves of the meninges.

Wilder [63] believes the vessels of the brain and meninges are dilated. Ocular headaches are due, according to Wilder (1) to exposure to bright light in cases of hypersensitiveness or irritation of the retina, which may be a precursor of retinitis, and to the frequent presence of venous dilatation; (2) to strain of accommodation in near use and in prolonged distant vision, giving rise to "academy headache," as it is termed in London; (3) to muscular insufficiency.

Frequency.—To compute the relation and frequency of ocular headaches to headaches due to other causes would be an interesting but an almost impossible task.[64] To learn the frequency of headache as a symptom of ocular disorder is only to review the case-book of any oculist in active practice. My own records show 85 per cent. in persons under 45 years of age who are free from recognizable organic disease of every description, ocular or general. Hospital records would of course show a less percentage since hospital cases include more diseased conditions. Zimmerman's statistics of all classes of headaches caused by ocular defects[65] show 67.8 per cent. in private practice and 74.9 in hospital cases. Zimmerman explains the higher ratio by the large number among hospital cases who must use their eyes for close work many hours in the day, such as weavers, woollen-mill workers, etc. Ninety-six of his patients had monocular vision only (strabismus or enucleation of one eye), and of these forty had headaches.

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[63] Ocular Headaches, J. A. M. A., November 19, 1898.

[64] C. A. Wood states that 40 per cent. of all headaches, and 80 per cent. of frontal headaches, are due to eye-strain.

Diagnosis.—The headache is usually induced by the use of the eyes, both in near work and in prolonged effort to see clearly in the distance, and consequently the connection of the use of the eyes with the development of the exacerbation of the pain is to be determined. It must not be forgotten, however, that some individuals, particularly with chronic headache, trace no connection between reading or other close work and the headache. In such cases it is practically impossible to ascribe the cause to the eyes until after the therapeutic or optical test is applied. The former consists of the instillation of a cycloplegic for a few days or the abolition of the accommodation. The latter, of the continuous wearing of the correction of any error of refraction that may have been found under paralysis of accommodation. Yet it must be remembered that the presence of muscular anomalies of sufficient degree to become a factor in the causation of headache vitiates these tests, for it is not uncommon to find that headache persists or is increased because of the participation of the extraocular muscles in the visual act. This is shown in cases of functional internal hyperopic squint. For some days after the initiation of the use of a cycloplegic the degree of squint may be higher than before its use. The innervation of the muscles of convergence is not relaxed because of the temporary failure of the ciliary muscle to respond to its innervation. The diagnosis is simplified by the discovery of ocular strain induced by hyperopia, myopia, or astigmatism, as shown by lowered acuity of vision, by the ophthalmoscope, ophthalmometer, retinoscope, and test lenses, or by imbalance of the ocular muscles, as revealed by the Maddox rod, cobalt glass and diplopia test, and the proportionately increased tension of the muscles of adduction and abduction or sursumduction.

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[65] New York Medical Journal and Philadelphia Medical Journal, November 21, 1903.

In every case knowledge of the functional activity of other organs of the body contributes materially to the accuracy of the diagnosis. Organic disease of the nervous system, abdominal organs, digestive tract, syphilis, gout, rheumatism, and other general affections should be excluded.

Simulating ocular headaches more closely than the other varieties are those caused by nasal disease. Here the pain is frontal, more often dull than sharp. Disease of the nose is, in many cases, excluded by examination of its cavities. But Ewing[66] describes a form of headache of nasal origin, characterized by neuralgia of the nasal and ethmoid nerves and tenderness on pressure on the inner upper angle of the orbit of one side, rarely both, in which the inflammation is not confined to the frontal sinus. Rhinoscopic examination revealed in his case no signs of the trouble. The headache is dull, persistent, located in the frontal region and generally worse in the morning. The cause of the headache is referred to the eyes, because the pain is greatly aggravated by attempts at reading, and attacks of conjunctivitis and blepharitis are frequent. The diagnostic features are tenderness, limited to one orbit or one nasal bone, and asthenopia.

The near work includes all the ordinary occupations—reading, writing, sewing, weaving, reading music, and amusements, such as card-playing. Ocular headache is quite common among card-players who have entered the presbyopic age, as the games are usually played by artificial light and for several consecutive hours. The asthenopia is readily explained by the illumination of the room, the glare of the light reflected from the cards, and the accommodative strain induced by demand for accurate vision at distances varying from one-third of a metre to one and one-half metres, and, in some cases, by the necessity for constant and vigorous mental action.

Presbyopia is frequently a cause of headache. The crystalline lens has become hard and inelastic, its layers of fibres gliding on one another to an exceedingly limited degree while the innervation to the ciliary muscle remains practically unchanged. The contraction of the muscle in response to the innervation is also almost as strong as early in life.

Ocular strain in the continuous effort to see clearly in the distance, such as the preacher in the pulpit and the actor on the stage, is a frequent cause of headache and other forms of asthenopia. The peculiarity of this headache is its occurrence some hours after the strain is over. "Panorama" headache, or car-sickness, is less to be attributed to idiosyncrasy of individuals than the reflex of disordered ocular function, brought about by the effort to fix distant and rapidly passing objects. The constant change of focus and line of direction of the visual axes and also the rapidly changing retinal image entail too much action of the accommodation, of the extraocular muscles, and of the retina and afferent visual impulses.

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[66] Trans. Amer. Ophthal. Soc., 1901.

Character.—The pain is sharp and neuralgic or dull and heavy, varying according to the temperament of the individual, the degree of the error of refraction, the length of time during which the near use of the eyes is demanded, age, and the presence of muscular anomaly. The first exceeds in importance, as a cause of intensity of pain, all other causes, and demonstrates that the cause of the trouble is not only ocular anomaly, but the combination of an ocular anomaly with a susceptible impressionable disposition. Low defects in subjects enfeebled by disease or whose power of resistance is weakened by suffering or mental distress may give rise to intense and constant headache, while in healthy and strong individuals equal defects, whether of refraction or muscular imbalance, are overcome or held in abeyance without any response whatever of the nervous system. In H. and HAs. of between 1 and 2 D. the pain is apt to be constant during the working hours, and is aggravated by close work. In mixed astigmatism of moderate degree, the headache may be constant and associated with nausea and vomiting and not infrequently with symptoms of some nervous disorder. Near work is either impossible or accomplished only at the expense of intense suffering, and, if persisted in, results in chronic invalidism. The so-called neuralgia and migraine attributed by medical men and laymen to malaria, rheumatism, anæmia, and other general and vague diseases, is, in the majority of cases, only an expression of a disordered ocular function. For example: G. L. Walton, *Boston Medical and Surgical Journal,* has made an interesting study of migraine among individuals blind from infancy and others who later in life became partly blind.

He found among 41 persons blind from infancy 80 per cent. had never had headache.

Among 18 cases becoming completely blind in life only 5 were free from headache.

Among 100 persons in good health only 31 per cent. had never suffered from headache. Seventeen had typical migraine (about one in six individuals), a marked contrast to the absence of typical migraine in the 41 cases of congenital and complete blindness. He believes that the explanation is to be sought in the frequency of eye-strain among those who have their sight, particularly among the partially blind, and its absence in the totally blind.

The neuralgia may be limited to one side of the face or may be bilateral. The supraorbital nerves are tender on pressure, or even painful spots located in the scalp simulating supraorbital neuritis may be present, and yet the cause be an ocular anomaly.

The ocular headache assumes many phases. It cannot be certainly diagnosticated without an ocular examination and proper correction, but it is safe to predict in any given case that the combination of H. and HAs., or esophoria with an average nervous susceptibility, will be the cause of headache, which will be temporarily relieved by rest and permanently relieved by well adjusted correction, worn constantly.

While headache is the most common form of asthenopia, other reflexes not so directly referable to the use of the eyes are worthy of mention,—for instance, migraine, neurasthenia, insomnia, restlessness, nervousness, enuresis, urticaria, depression of spirits or mental apathy. Examples of each of these affections and others are found in medical literature. Their diagnosis and treatment is identical with that of ocular headache, indigestion, and other reflexes of eye-strain.

Treatment.—The indications are twofold: first, the correction of the error of refraction by glasses and the muscular anomaly by appropriate remedies; second, hygienic and medical measures that will reduce the susceptibility of the nervous system, regulate the habits and modes of life, both as they pertain to the general health and more particularly to the ocular function. Ametropia may be corrected in individuals under 45 years of age who have hyperopic astigmatism only by learning the static refraction under paralysis of accommodation. The cycloplegic used is immaterial, provided it abolishes temporarily the contractility of the ciliary muscle. In myopes of more than 4 D. or 5 D. of myopia, cycloplegia is seldom necessary since such persons rarely suffer from accommodative headache and the ciliary muscle cannot conceal the optical defect. After the age of 45, cycloplegics are advisable only exceptionally, since the true refraction of the eye is manifest.

The eyes are not exclusive factors in the production of headache, but no diagnosis is complete which ignores them.

SPASMODIC MOVEMENTS OF THE HEAD OTHER THAN CHOREIFORM.

This affection may be due to organic disease of the cerebrum or of the meninges, to unnatural positions of the head in consequence of paralytic or functional imbalance of the ocular muscles, to traumatism of the neck or trunk, posture incident to some forms of chronic invalidism, rheumatism, and, possibly, to other deformities by which the physical equilibrium is disturbed. In the adult, head *shaking* is a symptom of paralysis agitans, disseminated sclerosis, general paralysis of the insane, aneurism and abscess of the brain. Head *tilting* is associated with functional and organic ocular muscle defects, oblique non-compensating bilateral astigmatism, traumatic or diseased contraction and paralysis of the neck muscles on one side, and to disease of the cerebrospinal system. In infants, involuntary rapid oscillation or other movements of the head, variously designated as head shaking, eclampsia nutans, head banging, salaam, convulsions of infancy, head bowing, and nodding convulsions, is accredited to arrested development of the nervous system, and is found in association with rickets, epilepsy, impaired intellect, and physical deformities. " Sometimes head shaking in infants is a form of half-involuntary movements, twitchings, antics, and grimaces to which children are subject. In this case there is no fear of epilepsy, and recovery is the rule." [67]

The position of the head assumed in compensation of heterophoria, heterotropia, and errors of refraction, is independent of central lesions and is purely voluntary. It is not in any sense a symptom of disease or a disease itself. It resembles the habit spasm or contractions of the facial muscles common in individuals in whom the visual act is accomplished only by undue muscular strain. The direction of the tilting of the head characterizes to a certain extent the variety of defect. Thus, in paralysis of the externus of the right eye, the eye is deviated to the left and the face is rotated to the right in order to bring the visual line of the right eye parallel to that of the left. In paralysis of the motor oculi of the right eye the right cornea is deviated towards the temple and the face is turned to the left; in paralysis of the elevators of the cornea the chin is raised in order to bring the depressed axis of the paralyzed eye in line with that of the sound eye; in paralysis of the depressors the head is tilted forward with the chin receding. Bard [68] states that ocular deviation and rotation of the head after hemiplegia is due to active movements of the muscular groups of the unaffected side automatically governed by the unilateral action of central perceptions, or simply from the reflex power of the sensorimotor centres. Sight predominates, but other senses take part, particularly hearing and those of locality and equilibrium. But slightly influenced by the seat of the lesion, it is affected by the gravity, extent, suddenness and intensity of the shock and by the degree of coma. Both ocular deviation and head rotation disappear either on recovery of the affected hemisphere or extension of the lesion to the other side of the brain. In heterophoria—latent squint—tilting of the head or face is less noticeable than in heterotropia (manifest squint), since in the former there is no actual deviation of the visual axes. The tension of the weaker set of muscles is partly substituted by contraction of the occipito-frontalis, corrugator supercilii, or the orbicularis palpebrarum. The undue stimulation of the facial muscles may be extended to include adjacent muscles of the neck or back, by the contraction of which the erect posture of the head or trunk is modified. Lewis[69] reports a case of pseudotorticollis in a girl eight years old. She complained of frequent headache during the winter school session. Previously a good scholar, she became dull, inattentive, irritable, and thin. During the summer vacation she gained in weight and the headaches were less, but improvement was cut short by resumption of school studies. The symptoms returned, aggravated by marginal blepharitis and lachrymation. She held her head in a depressed position downwards to the right side, so that the eyes when fixing an object directly in front were directed upwards and to the left, the upper lids covering the irides almost down to the pupillary margins. Slow rhythmic swaying of the head when looking at the test letters was a constant symptom. There was no stiffness of the cervical muscles. The oblique position of the head was maintained in the endeavor to overcome the imperfect vision due to the need of mixed cylinders (R. $-75 = +2°$ ax. 100) right hyperphoria of $3\frac{1}{2}°$ and esophoria 2°. In testing the near vision the same rhythmic swaying was noticed. Gould[70] relates an interesting and instructive case of torticollis and spinal curvature due to eye-strain (from astigmatism). He says the case should be interesting to the general physician, orthopedic surgeon, and oculist, because the patient was of tuberculous parentage, and it was feared that she would develop tuberculosis. The symptoms simulating tuberculosis were soon found to be caused by the false position of the head, that in turn by wry-neck which had caused spinal curvature. The patient, a girl of nineteen, complained of severe and protracted headache, sore eyes, and pain in the eyeballs. The erect position was resumed and all the symptoms both of eye-strain and tuberculosis disappeared after correction of compound hyperopic astigmatism. Without her glasses the head takes its former abnormal position, the right shoulder drops, the back becomes more humped, etc. The regained health is primarily due to the glasses and secondarily to the proper position of the body, increased lung capacity, and better oxygenation of the blood. A second case of torticollis was a woman of 25, with high degree of astigmatism in both eyes, axes 170°, a defect which would not allow binocular vision with the head in its normal position. Gould alludes briefly to other cases, whose histories are confirmative of the causation of torticollis and spinal curvature by ocular defects and the cure of those affections by relief of the eye-strain.

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[67] Samuel Gee, St. Barthol. Hosp. Rep., 1886.

[68] Semaine Médicale, January 13, 1904.

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[69] Oph. Rec., 1903, No. 12.

[70] American Medicine, March 25, 1904.

Wadsworth[71] reports a case of *spastic torticollis* in a boy of fourteen which had persisted since infancy. The boy was healthy and had good muscular development. He sat or stood with his head tipped to the right and inclined considerably forward, his right shoulder lower than the left and the spine laterally curved, with convexity to the left. The ocular excursions were good in all directions. In ordinary positions, with fixations for near or distance, the left eye stood out about one degree higher than the right. Only when the eyes were turned strongly down and to the left, while at the same time the head was inclined forward and to the right, did he apparently get binocular fixation. The superior rectus was divided, with the result that the body has been much straighter ever since, and is still improving.

Walton[72] reports the case of a boy 12 years of age who had a general chorea, head jerking being the most marked symptom. The child had an esophoria of 6°. His internal recti were tenotomized, and, when seen two weeks after the operation. the twitchings had almost entirely ceased.

Recurrence of head shaking, head tilting, and torticollis may be expected upon the return of the muscular insufficiencies. Should they relapse following a change in the refraction of the eyes, especially in the axis of the astigmatism, they may be again relieved by restoration of normal ocular conditions.

Posey[73] classifies the head movements into (1) physiologic, or "the many and varied movements which the head makes through the medium of the neck muscles;" (2) abnormal independent, but simultaneous, or the accidentally simultaneous, occurrence of nystagmus and head tremor; (3) compensatory, or a vicarious position of the head assumed to compensate for some imperfect action of one or more of the extra-ocular muscles, and (4) related, but not compensatory, or those anomalous movements of both head and eyes, the result of central lesion. Under the third heading he refers to cases reported by Wadsworth, Risley, and Wilder, and adds one case of his own. Under the fourth class he describes a new case.

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[71] Trans. Amer. Ophthl. Soc., vol. v.

[72] Boston Medical and Surgical Journal, 1892.

[73] Associated Movements of the Head and Eyes, Journal of the American Medical Association, November 29, 1902.

Head Bowing of Infants.—This affection is essentially different in its pathology and etiology from the head movings or tiltings of adults, symptomatic or ocular disturbances. In infants the affection is attributable to organic changes in the brain or its membranes, the nature of which is unknown. Another and entirely distinct form of eye, head, and hand movements, which should not be confounded with the true eclampsia nutans, is that sometimes seen in children whose vision is reduced to the perception of light from ophthalmia neonatorum, or congenital cataract. In the latter affection, nystagmus is invariably present. The head is moved slowly, horizontally, and vertically and one or both hands are held between the eyes and the light and rapidly moved within a narrow area. In the former the infants are rhachitic, hydrocephalic, strumous, or tubercular. William Nestham,[74] who seems to have studied this affection with great thoroughness, says, "There is evidently in this malady a family alliance with epilepsy, and hence, as has been demonstrated by the pregoing cases, it often passes into epilepsy or other form of infantile convulsions. Tetanoid symptoms also sometimes occur during its progress." The disease has been attributed to dentition, irritation of spinal nerves from the presence of worms or other irritating matter in the body and also to atmospheric changes. His conception of the morbid action is that the essential character of this malady is inflammatory action in the weak or strumous, which in all probability commences in the membranes investing the medulla oblongata. It is early extended to the membranes covering the base of the brain, and is succeeded by disease of the vessels supplying and nourishing the organ itself. This condition is then followed by exudations of lymph or serum, the locality of these effusions being determined by the constitution and local circumstances, and, as a consequence of these exudations, paralysis is produced from pressure. The regular nutrition of the brain is interrupted, and, in the more aggravated cases, the organ becomes so deteriorated as to lose all power of carrying on the intellectual functions.

The rapidity and frequency of the head movements differ in different children, and in the same child at different times. The movements may recur at intervals of months, each spasm continuing a few min-

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[74] London Medical Record, May 14, vol. xi. 1881.

[illegible] for several hours. The head [illegible] because the child is not [illegible] the upright position, [illegible] changed for [illegible] as a pillow. The [illegible] movements, or may [illegible] Dr. Stephen Mac- [illegible] of more or less [illegible] of a nodding of [illegible] patient. In each there [illegible] confined to the [illegible] by restraining [illegible] during sleep.

[illegible] of the published cases [illegible] began between four and [illegible] the period during which [illegible] movements of the eyes [illegible] of age the infant has [illegible] together, and [illegible] little skill in following [illegible] have been described [illegible] but no details [illegible] twentieth month. [illegible] symptoms may still be [illegible]

[illegible] When the affection [illegible] phimosis, [illegible] upon removal of [illegible] not too seriously [illegible]

[illegible] nystagmus follow- [illegible] that the pupils [illegible] were healthy [illegible] was almost [illegible] in combination with [illegible] about six years old.

[illegible]

The infants who survive the disease of the cerebrospinal system of which the head nodding is a symptom, are usually permanently enfeebled, either physically or mentally. The ophthalmoscopic findings are, as a rule, negative, but should optic neuritis or optic nerve atrophy be a complication the prognosis is unfavorably affected.

Treatment.—The principle upon which the treatment is based varies according to the most probable cause of the affection. Should spinal irritation be suspected, calomel to keep the secretions in order and tonics to support the strength are indicated. If dental irritation be present, thorough and repeated lancing of the gums is called for. The avoidance of everything that tends to increase cerebral action should be a part of the treatment. The bromides are useful. Nourishing diet at regular and frequent intervals, abundance of fresh air and sunlight, well ventilated sleeping-rooms, suitable clothing, and other hygienic measures, constitute an essential part of the treatment.

CHAPTER XIX.

EXOPHTHALMIC GOITRE.

BY WILLIAM CAMPBELL POSEY, M.D.

EXOPHTHALMIC GOITRE, Graves's disease, or, as it is designated in Europe, Basedow's disease, is an affection characterized in most cases by a group of symptoms which consist of increased frequency of the pulse, enlargement of the thyroid gland, the presence of certain characteristic ocular changes, a fine tremor, and nervous irritability. The disease is essentially chronic in its course and is of unknown origin, though probably dependent upon some disturbance of the central nervous system induced primarily by changes in the thyroid gland.

Although others had practically described the disease, in reports of cases which were undoubtedly true instances of exophthalmic goitre, the honor of recognition of the symptoms as belonging to a distinct and separate morbid process should be accorded to Graves (1835), though it was not until five years later (1840) that Basedow, ignorant of Graves's discovery, fully described the disease with its cardinal symptoms.

SYMPTOMATOLOGY.

All the five primary symptoms which have just been mentioned are not always present at the same time in every case, and one or more may be absent during the entire course of the disease; the tachycardia and the nervous irritability appear, however, to be present in all cases. In addition to these cardinal symptoms, there are other secondary symptoms which are quite frequently present; these include changes in the alimentary canal, in the respiratory and genito-urinary organs, and in the skin.

Vascular System.—**Increased Frequency of the Pulse; Tachycardia.**—The most constant and usually the first symptom of exophthalmic goitre is a marked increase in the force and frequency of the contraction of the heart; the pulse may beat as rapidly as 120 to 160 times in the minute, and even 200 beats have been recorded; it rarely sinks to less than 100. The action of the heart is often irregular and may be at times intermittent; it is usually perceptible to the patient, especially [illegible] en slightly increased by exertion or excitement. The cardiac impulse is strong and extends over a larger area than is normal. In the early stages the sounds of the heart are usually unaffected, but later, as a consequence of the prolonged increased activity in the cardiac action, the sounds become louder than normal, and Gowers [1] is authority for the statement that they may be so intense at times as to be heard as far as four feet from the patient.

Murmurs present in the early stages of the disease are usually of anæmic origin, but later actual murmurs from faulty valvular action, as a consequence of hypertrophy and dilatation of the heart, may arise. Pulsation in the arteries is common and can be readily seen and felt in the large arteries at the root of the neck and even in the peripheral vessels; in thin subjects pulsation can be demonstrated in the abdominal aorta.

Nervous System.—**Tremor and Nervous Irritability.**—Tremor is probably present in all cases at some time or other; though usually an early symptom, it may not manifest itself until after other symptoms have appeared. It is most marked in the extremities, particularly the upper, though it may, however, extend over the entire body. The character of the tremor varies, but is usually fine and regular. In cases where the exophthalmus and goitre have been limited to one side of the body, the tremor may be unilateral also. It is best elicited by requesting the patient to stretch out the hands with the palms directed downward, when, if too slight to be seen, by laying the palm of the hand lightly upon that of the patient, the observer will note that the trembling is not in the fingers alone, but that the entire hand moves, this being occasioned by a spasmodic action of the flexor and extensor muscles of the wrist. The tremor is increased by excitement or by attempting to perform actions with extreme care, and resembles the tremor of fatigue and weakness in being a little more frequent in time than that of paralysis agitans or of disseminated sclerosis (page 908).

In nearly all cases of Graves's disease there is a slight mental change which usually manifests itself in an unwonted irritability and restlessness. There may be times when the patient regains his usual spirits, but he soon lapses again into a peevish and depressed state. In other cases the mental derangement takes the form of a nervous expectancy, and the patient is in a flutter of excitement about trifles. Nervous symptoms, as a rule, develop in individuals who are predisposed by a neurotic ancestry, but they may appear in subjects who were quiet and unexcitable or even phlegmatic before the disease manifests itself.

In addition to this general nervousness, there are not infrequently other nervous symptoms which are of a functional rather than of an organic nature. Hysteria is often present and may occur under various forms, though some variety of anæsthesia or paralysis is the most common. Many of the convulsions which occur in the course of exophthalmic goitre are of an hysterical origin. Headache and migraine in its typical form, neuralgia, dizziness, ringing in the ears, and other similar neuroses have also been met with.

Actual insanity may supervene, taking the form of melancholia and mania or, more rarely, that of recurrent mania and general paralysis. The prognosis is always rendered grave by this complication, as death ensues in some cases.

Paralyses of cranial nerves are not very unusual, probably from nuclear involvement. Symptoms of tabes have been observed in a number of cases; paraplegia may manifest itself, either with flaccidity or rigidity of the legs, and Charcot[2] and Maude[3] have observed an inability to stand or walk, while the legs can be freely moved when lying down (*astasia-abasia*), as an early symptom.

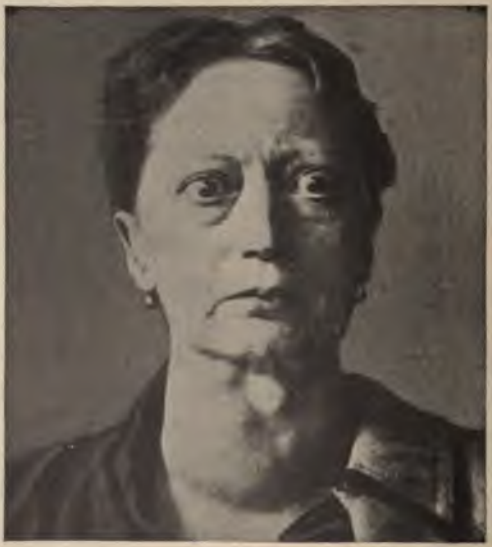

FIG. 1.—A case of exophthalmic goitre. (From Wilbrand and Saenger.)

Rarer complications include chorea, tetany, chronic muscular atrophy, paralysis agitans, Thomsen's disease, rheumatoid arthritis, and osteomalacia.

The Enlargement of the Thyroid Gland.—This usually occurs simultaneously with the exophthalmus. It may, however, exist for some years before any of the other symptoms of exophthalmic goitre appear. Of eighty-seven cases seen by Murray, in which either one or two symptoms appeared simultaneously before the others, an enlargement of the thyroid gland was the first symptom of the disease in forty-three. Though not so constant a symptom as the tremor, it is present in most of the cases, Murray finding enlargement of the gland absent during some period of the disease in but three out of one hundred and twenty cases recorded by him (2½ per cent.). Griffith[4] failed to find an enlargement at the time of examination in thirteen out of thirty cases. The gland, as a rule, is but moderately enlarged, and rarely attains the size of parenchymatous goitres. The enlargement is usually symmetrical upon both sides, though it frequently happens, especially when the exophthalmus is unilateral, that one lobe is more enlarged than the other; when this is the case the right lobe is usually the larger.

The vessels of the growth are usually much dilated, and pulsation of the entire gland is generally palpable and visible as well, especially when the heart's action has been increased by excitement. A thrill can generally be felt on the gland, and the ear will detect a loud systolic murmur or often a *bruit de diable.* Guttman considers a double murmur to be pathognomonic.

THE OCULAR SYMPTOMS.

These constitute a very striking feature of the disease, and though none of them, when existing alone, can be considered to be pathognomonic of exophthalmic goitre, the association of one or more of the most significant with but one of the other cardinal symptoms of the disease is sufficient to establish the diagnosis of exophthalmic goitre. They may best be studied in the following order:

The Exophthalmus.—Actual protrusion is not always easy to determine, especially as the development of the exophthalmus is usually gradual, and the clinician will frequently be compelled to rely upon the observation of the patient or his family and friends or to have recourse to photographs taken earlier in life before it can be definitely asserted that an actual protrusion of the globe has occurred. Care must also be taken to differentiate unusual prominence of the eyeball due to increased length in the axis of the globe (myopia or anterior staphyloma) or to shallow orbits, from true exophthalmus. The degree of the exophthalmus varies greatly in different cases, in some being so slight that it occasions a mere staring expression, while in others it is so pronounced that the lids cannot be completely closed over the globe; in extreme cases the eyeball may even be dislocated in advance of the lids (Pain.).[5]

The degree of the exophthalmus may change from day to day, the protrusion being made greater by whatever increases the heart's action. Its progress also is not always uniform, for there are frequent periods when the globe appears to remain stationary. In some cases the exophthalmus has appeared only under special circumstances; thus De Mussy found it in a case only during choreic attacks and Savage in another case at the time of menstruation alone, when there were symptoms of mental disturbance.

The eyeballs can be pushed easily back into the orbits, though they come rapidly forward as soon as the pressure is relieved. The protrusion is generally directly forwards, and both eyes are, as a rule, similarly affected, though the exophthalmus may be limited to one eye.

A case of this nature was presented by Dr. Swindells and the writer before the College of Physicians of Philadelphia. In this patient, a female, aged 22, the left eye became suddenly proptosed two years previously following a fright. She had been nervous for some years and had suffered from irregular menstruation. Her mother was neurotic and had goitre, but no exophthalmus. A maternal grandmother had died of cancer of the throat. The exophthalmus in the left eye was of moderate degree and the right eye was normal. All lid signs were absent except infrequent winking. The gland was bilaterally enlarged, though the right side was the larger. Marked improvement in the general and local condition followed a strict rest cure, galvanism, and syrup of hydriodic acid.

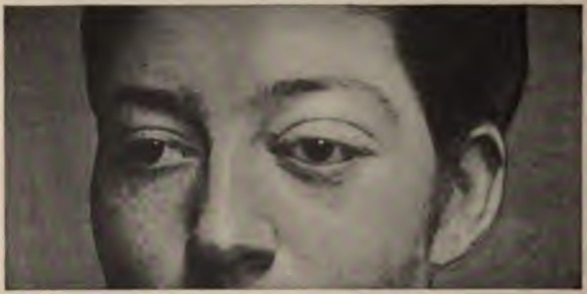

FIG. 2.—Unilateral exophthalmus.

A comprehensive monograph upon the subject of unilateral exophthalmus was contributed by Fridenburg in 1895. This author reported one case in his own practice and one from Jacoby's, and gave [6] in addition the notes of ten additional cases which he had collected from the literature. Since the publication of this article additional cases have been reported by Sichels,[7] Völkels,[8] Praël,[9] Mauthner,[10] Mooren,[11] Rösner,[12] Emmert,[13] De Giovanni,[14] Barella,[15] Hitscham,[16] Mendel,[17] Trousseau,[18] and Bistis.[19]

A number of observers have remarked that in many of these cases the enlargement of the thyroid was limited to one side of the gland, and that upon the side on which the eye was proptosed. The majority of the cases, however, show no such association, and no special significance can be attached to the occurrence. Sattler [20] explains unilateral exophthalmus by a disturbance of the vasomotor centres of the orbit of the affected side.

The Various Lid Signs.—**The Graefe Sign.**—This sign, which consists in the impairment of the consensual downward movement of the upper lid with that of the globe (see page 351), was described by Von Graefe [21] in 1864. Though present in many cases of exophthalmic goitre, it cannot be considered to be pathognomonic of this disease, for it may not infrequently be demonstrated upon the eyes of healthy individuals. Thus, Sharkey [22] observed it twelve times among six hundred and thirteen cases of all kinds of disease, exophthalmic goitre excluded. The same author found, too, that a large proportion of healthy people can voluntarily produce this sign and the one next to be described—the Dalrymple—by staring at an object in front of them. The writer has frequently noticed that when patients are first made to fix a near object in testing the balance of the extraocular muscles, the palpebral fissure

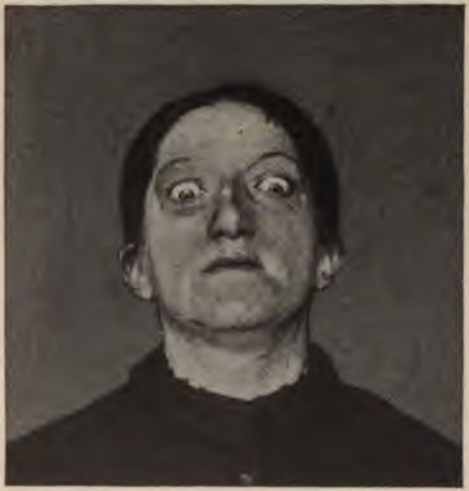

FIG. 2.—Graefe's lid sign in exophthalmic goitre. (From Wilbrand and Saenger.)

frequently widens and the Graefe sign can be demonstrated. In such cases, however, in a few moments, especially after several trials have been made, the fissures assume their normal width, and the normal relationship between the movement of the lids and the globe is regained, showing, in the opinion of the writer, that the anomalous behavior of the lids and globe was due to a temporary incoördination produced by a confusion in the mind of the patient as to what particular demand was being made upon him. Hughlings Jackson,[23] Wilbrand and Saenger,[24] and Flatan[25] also have observed the symptom in healthy individuals. Raymond[26] noted it in two cases of Thomsen's disease. Kocher[27] and Jessup have shown that similar phenomena, but to a lesser degree, may be produced by instilling cocaine into the conjunctiva, and deemed the sign to be occasioned in these cases by a direct stimulation of the sympathetic nerve-endings, and a consequent contraction of the orbital muscles supplied by these nerves. As further evidence that the Graefe sign is not pathognomonic of exophthalmic goitre, it may be remarked that there are many cases of this disease in which it does not occur at all; thus, Mannheim[28] observed it but eighteen times in forty-one cases, and Russell Reynolds[29] in but four out of forty-nine. Pässler[30] saw it but nine times among fifty-eight cases; Bruns[31] ten times out of forty-two cases; Flatan[25] sixteen times among thirty cases, and Murray[32] thirty-six times among ninety-one cases.

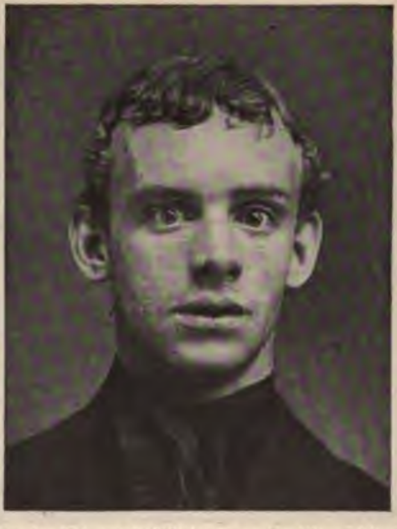

FIG. 4.—Dalrymple lid sign in a healthy subject.

An impaired movement similar to that observed in the upper lids has been noted upon several occasions in the ascent of the lower lids also, but as Maude has pointed out, this is never so marked as in the upper lid, on account of the greater range in the downward rotation of the globes. The Graefe sign occurs quite independently of the degree of the exophthalmus, and may disappear during the course of the disease and reappear again without the exophthalmus having changed its degree.

The Dalrymple Sign.*—This refers to the peculiar staring expression which patients with exophthalmic goitre present. It is occasioned by an abnormal widening of the palpebral fissure, due to retraction of the eyelids, and gives rise to one of the most striking symptoms of the disease (Fig. 4). (Compare Page 350).

The Stellwag Sign.—This is the name applied to the infrequency and incompleteness with which the act of winking is performed. It is a very marked symptom in some cases and may lead to corneal ulceration.

Too frequent nictitation has been observed in some cases; thus, Hitschmann[33] has reported a case where the act of winking was performed about thirty times a minute.

Both the Dalrymple and Stellwag signs may be confined to one eye, though they usually occur in both. They are more marked at times than at others, being increased by fatigue, excitement, or anxiety. They may each exist independently or in combination with one another or with exophthalmus and the Graefe sign. The figures of Wilbrand and Saenger[34] are of interest in this connection. Among thirty-nine cases of exophthalmic goitre seen by these authors, exophthalmus was present in twenty-seven cases, in twenty-three of which the exophthalmus was bilateral and in four unilateral. Among these twenty-three cases, exophthalmus, Graefe sign and Stellwag sign occurred simultaneously in five cases; exophthalmus, Graefe sign, and Stellwag sign occurred simultaneously in four cases in the same eye; exophthalmus, Graefe sign, and Stellwag sign in fourteen cases; exophthalmus, Graefe sign, and Stellwag sign in four cases; exophthalmus, Graefe sign, and Stellwag sign were absent in six cases; Graefe and Stellwag signs in one case; Stellwag sign alone in two cases; Graefe sign only in three cases.

Mannheim's[35] statistics show that among forty-one cases of exophthalmic goitre the Graefe, Dalrymple, and Stellwag signs existed at the same time in ten cases, and that six showed the Graefe sign without the other two, while the Graefe sign was absent and the two others present in four cases.

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* This sign is frequently included as part of the Stellwag sign, but the credit of its discovery should be given to Dalrymple, who first pointed it out twenty years before.

Despite these figures, however, it is probable that if carefully searched for, all three of the signs will be found at some stage of the disease, and while their presence cannot be considered to be pathognomonic of exophthalmic goitre, it is of great value in confirming other symptoms.

The Möbius Sign.—According to Möbius,[36] unilateral convergence is normal in exophthalmic goitre, but the force of binocular convergence is sensibly diminished, quite independent of any mechanical obstruction offered to the proper convergence of the globes by extreme exophthalmus or from any paralysis of the ocular muscles. This weakness does not occasion diplopia, but evidences itself in a troublesome asthenopia, which frequently leads the patient to consult an oculist for relief.

Paralysis of Extraocular Muscles.—In cases of marked exophthalmus there is frequently more or less restriction in the movements of excursion of the eyeballs, although, as the degree of this limitation is about the same in both eyes, the patient suffers but little upon that account. There may, however, be actual palsy of the extraocular muscles.

Interesting monographs upon this subject have been written by Ballet,[37] Liebrecht,[38] Buschan,[39] Mannheim,[40] and Möbius.[41] It would appear, from the cases reported and cited by these authors, that the paralysis may affect a single muscle or group of muscles of one eye, or even all the extrinsic muscles of one eye; and that occasionally one or more muscles of both eyes are simultaneously affected. Palsy of the ciliary muscle or iris is not recorded, nor of any of the associated movements of the eyes, with the exception of reports by Schmidt-Rimpler [42] and Vossius [43] of cases of paralysis of convergence. Palsy of the levator palpebræ superioris in conjunction with palsy of the other branches of the third nerve is rare, but is rather more common without such association. The palsy may come on early in the course of the disease, but in most cases it is a late manifestation; though usually persistent, it may be but a transient condition, the muscles regaining their full strength.

The palsy may occur without paralysis of other cranial nerves, or it may be associated with a similar affection of one or more of them; thus, Bristowe [44] has recorded the case of a young man who developed the symptoms of ophthalmoplegia externa three years after the ordinary symptoms of Graves's disease had manifested themselves. There was also right hemianæsthesia, with color-blindness and loss of taste and smell on the same side, some palpitation and dyspnœa with headache and sickness. After a period of some months, epileptic fits came on, and later on hemorrhages from both ears. Death finally occurred from bronchitis. Autopsy was negative.

Similarly, in a case reported by Warner [45] there was binocular external ophthalmoplegia with palsy of the facial and trigeminal nerves. Jendrassik [46] saw paresis of the muscles of mastication, in addition to palsy of the rotary muscles of both globes, as well as most of the muscles of the face and palate. Of interest in this connection also are four cases reported by Ballet; [37] the first, the subject of severe hysteria, in addition to ophthalmoplegia externa, presented a number of bulbar symptoms,—*i.e.*, palsy of both facials and paresis of the hypoglossus. In the second, also the subject of severe hysteria, there was right-sided hemianæsthesia and loss of smell and taste on the same side. Autopsy showed no visible change in the central nervous system. In a third case, in addition to palsy of both external rectus muscles, there was right-sided hemiplegia and hemianæsthesia, with epileptic attacks. The fourth case was an instance of ophthalmoplegia externa with paresis of the facial and hypoglossus. Chevalier [47] saw palsy of the externi and oblique muscles associated with disturbance in the supply of the fifth and seventh nerves and of speech.

In a case of Maude's [48] the lesion seemed to spread over the entire region of the nuclei of the extraocular muscles and those neighboring upon them; paresis of the external rectus, and probably of the superior oblique, being preceded by palsy of the facial, and quickly followed by general ophthalmoplegia.

Stellwag's [49] case was one of paralysis of the lateral rotators (the ocular axes being parallel and convergence undisturbed), and later palsy of both externi with convergent strabismus and diplopia developed with transient anæsthesia and restricted movement of the left half of the upper legs.

In a case reported by Féréol,[50] of a man 41 years old, six months after the appearance of the initial symptoms of exophthalmic goitre there were headache, vomiting and dizziness, tremor, with disturbed gait, and a disposition to face to the right; then diplopia, the result of right-sided trochlearis palsy. There was also right-sided diminution of motion and hyperalgesia, while on the left side there was a diminution in sensation of pain.

Quite recently Voss,[51] of St. Petersburg, has reported two additional cases of palsy of the extraocular muscles in exophthalmic goitre. The palsy in the first case had affected the right superior and external rectus muscles of the right eye and the inferior and external rectus muscles of the left eye. There was also a partial palsy of the right facial nerve, with some cerebral symptoms.

The second case exhibited multiple lesions in the muscles of both eyes, the superior rectus muscle of the right eye being particularly affected.

Voss refers to a case of Bartholow's where there was ptosis in association with gangrene and neuritis of the lower extremities.

Dyson [52] reported the case of a man with the symptoms of incipient Graves's disease, in which, in addition to slight exophthalmus and the Graefe lid sign, there was slight internal convergent strabismus and dilatation of the pupil of the left eye. The patient was a chronic alcoholic.

Bruns [53] saw ptosis on the left side, with palsy of the superior recti in both eyes, with the cardinal symptoms of exophthalmic goitre, but as the patient was the subject of a chronic nephritis, it was thought that the nuclear lesion might be referable to this source and not to Graves's disease.

Wilbrand and Saenger [54] report a case with slight ptosis on the left side, with retraction of the upper lid, with Graefe lid sign, upon the right side. This was explained by a bilateral affection of the nuclei of the levator,—*i.e.*, an irritation of the levator on the right and a palsy on the left side.

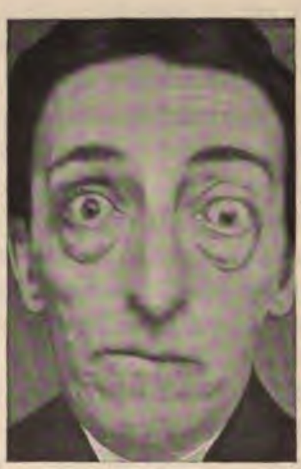


FIG. 5.—Paralysis of eye muscles in exophthalmic goitre.

De Giovanni [55] has reported a case of unilateral exophthalmus as a consequence of palsy of the extraocular muscles, especially of the rectus superior, in which goitre and other classical symptoms of Graves's disease were present.

The accompanying figure is that of a patient presented by the writer before the College of Physicians of Philadelphia, in whom there were all the cardinal signs of exophthalmic goitre, and, in addition, paralysis of all the extraocular muscles of the left eye, with the exception of the superior oblique.

In addition to these cases of more or less complete ophthalmoplegia externa, others, though more uncommon, of palsy of a single eye-muscle have been reported. These have been grouped by Buschan,[56] as follows:

(1) *Cases in which diplopia was complained of, but the muscles at fault not ascertained:* Trousseau Smith, Chvostek, Stellwag, Impaccianti, Strümpell, F. Müller, Lang-Pringle. (2) *Cases of palsy of the rectus internus:* West, Homen, Cohen, Westedt, and Romberg (rectus interni of both eyes); Schoch and Rothmann (rectus internus and externus). (3) *Cases of palsy of the rectus externus:* Kurella, Mackenzie (several cases), Eulenburg, Stellwag, Makeig Jones, Liebrecht, Dyson, Ballet, Cohen. (4) *Cases of palsy of the rectus superior:* Sollier, Fischer-Degranges, Kahler, Roth, Schlesinger, Chvostek. (5) *Cases of palsy of the trochlearis:* Féréol. (6) *Cases of palsy of the levator palpebræ of both eyes:* Maude, West.

Changes in the Cornea.—Notwithstanding the exposure to which the eyeball is subjected by the widening of the palpebral fissure and by the extreme degree of exophthalmus in many instances, and to the lessening in its moisture by the infrequent winking, and, furthermore, by the areas of anæsthesia, which may be demonstrated in the cornea in some cases of exophthalmic goitre, ulceration of that membrane occurs but rarely, appearing to depend more upon individual predisposition than upon any of the foregoing factors, those who are feeble and cachectic as well as those in advanced years, being particularly liable to severe corneal ulceration. Corneal complications show a tendency to occur more frequently among men than women, which is the more striking, in view of the fact that exophthalmic goitre is far more common among females. This may possibly be explained by the greater severity with which the disease attacks men and also by the advanced age at which they are affected.

In cases where the cornea is but slightly diseased, its epithelium becomes thickened, giving a dull and lustreless appearance to that membrane. The conjunctival vessels are usually dilated, as a consequence of which the patient experiences sensations of more or less irritation and dryness of the eyes, especially after reading. In more severe cases one or more small yellowish-gray areas of infiltration appear under the epithelium. The epithelium over these areas is thrown into folds and finally breaks down into shallow ulcers. In other cases, where the exophthalmus is of extreme degree and the lids do not close and properly protect the cornea at night, the lower third of the cornea becomes covered with thick yellow crusts, beneath which ulcers form, and not infrequently necrosis of the cornea and perforation of the eyeball ensues. It should be remarked, however, that extensive ulceration may occur in individuals where there is but slight exophthalmus, and in cases even where there is a plentiful supply of tears, the process in such instances being similar to that observed in keratitis of a neuroparalytic origin, the so-called *keratitis xerotica.*

Anomalies of Lachrymation.—Berger [57] called attention to a number of cases in which he claims that epiphora was the initial symptom of exophthalmic goitre, and Vernean and Diaman Herger have reported similar cases. The first author regards the epiphora as a secretory neurosis of the lachrymal gland, being due to a reflex condition of sensory excitation of the sympathetic vasomotor fibres. Most authors, however, agree with Sattler in ascribing the epiphora to two factors: (1) The increased width of the palpebral fissure, whereby a larger portion of the conjunctiva is exposed to the action of the air than is usual, and thus, being irritated, excites reflexly an increased flow of tears, and (2) the interference with the proper sucking up of the tears by the lachrymal apparatus, as a consequence of the imperfect and infrequent winking and closing of the lids.

In cases where the disease is of long standing, there is frequently a diminution in the amount of tears, and an actual dryness of the eye ensues; this may be accredited to a lessening in the sensibility of the sensory corneal and conjunctival nerves, as the result of exposure and irritation, and to a corresponding lessening in the reflex secretion of tears. (Compare Page 344.)

Unusual Ocular Manifestations.—**Obliteration of the Sulcus Orbito-palpebralis Superior.**—In spite of the emaciation which is often seen in subjects of exophthalmus, the sulcus, which is usually deep under such circumstances, may be obliterated, and in its place a sac-like fulness of the lid may appear, which is occasioned by the increase in the contents of the orbit due to the fulness and stretching of the blood-vessels.

Œdema of the Lids.—Œdema of the lids is sometimes present; it may be an early symptom, or may not come on until after some years. Such swelling is often very persistent, and may remain after all the other symptoms have disappeared. Mackenzie [58] considered it to be somewhat akin to the swelling of myxœdema and to be quite independent of the exophthalmus.

A bag-like swelling of the under lid, with dilatation of the veins at the ciliary bord [illegible] ciated with severe pain in the forehead and over the vertex, has been described by Stellwag.[59] Vigouroux[60] found a false œdema of the lids to be of frequent occurrence. He considered this to be dependent upon paresis of the orbicularis, as he observed that when this muscle was made to contract by electric excitation, the swelling disappeared.

Tremor of the Lids.—Homën[61] has noticed a tremor in the upper lids when the eyes were closed, which differed from the tremor seen in the lids in other diseases of the nervous system and occasionally in health by the rapidity with which it manifested itself after the lids were closed, by its regularity and rhythm, and its extremely fine character. A similar tremor was noted by Liebrecht.[62] In this case the motion in the lid could be stopped after a few moments by pressure upon the supraorbital and facial nerves.

Nystagmus.—Cases of nystagmus have been reported by Bramwell[63] and Freund.[64] The latter author believes the movements in the eyeballs, although differing in rhythm, to be analogous to the tremor in the extremities.

Changes in the Fundus Oculi.—With the exception of a tortuosity and dilatation of the retinal veins which are present in some cases, changes in *the retinal vessels* are very uncommon. O. Becker[65] saw a dilatation of their walls and the presence of a pulse isochronous with the radial pulse in the retinal arteries in ten out of thirteen cases, but most observers have failed to substantiate these findings.

In like manner, changes in the *optic nerves* are uncommon, and the cases of double optic neuritis which have been recorded by Story[66] and Rieger and Forster[67] and the two cases of optic atrophy seen by Emmet[68] should be regarded as complications rather than as symptoms of the disease.

Refraction.—In thirty cases examined by him, Griffith[69] found the refraction to be normal in twenty-two instances. Of the remaining eight, four were myopic, one highly hypermetropic, and three astigmatic.

Field of Vision.—Notwithstanding the observations of Kast and Wilbrand,[70] who found concentric narrowing of the field of vision with or without impairment of the central vision or the color sense in subjects free from hysteria, but exhibiting more or less signs of cardiac palpitation, the visual fields in exophthalmic goitre, except in so far as they may be influenced by an accompanying hysterical condition, are normal (see page 638).

The Cutaneous System.—The changes in the integument may affect (*a*) **the vasomotor system**, occasioning at times the so-called "tache cerebral," œdema, and peripheral changes in temperature. Of these the "tache cerebral," the transient redness which follows even the slightest touch of the skin, is a not infrequent symptom of exophthalmic goitre, and was attributed by Trousseau[71] to a marked asthenia of the vaso-motor apparatus. Œdema may be either general or local. It occurs for the most part in debilitated subjects and is influenced by gravity, though there are other forms of circumscribed œdema probably akin to those observed in myxœdema. Increase in the bodily temperature has been noted not rarely, and may occur in all the stages of the disease without apparent cause. This actual increase in temperature is to be differentiated from the sensation of excessive heat, without increase in temperature, which is at times complained of and to which Charcot has assigned the name of thermophobia.

(*b*) **The glands,**—excessive sweating. Excessive sweating upon slight cause is one of the most frequent symptoms of the disease, and may be local, as, for example, when it is confined to the hands, or, what is more often the case, it may involve the entire surface of the body. It may be confined to one-half of the body. Diarrhœa is sometimes most troublesome, and is thought to be a kind of intestinal sweating.

(*c*) **The skin**—pigmentation. Pigmentation of the skin has been observed by a number of authors. In most cases it assumes a bronze tint, comparable to that in Addison's disease, though never so pronounced as in advanced cases of this affection. Vitiligo and urticaria eruption, and a double form of rash consisting of dusky papules like the eruption of measles in its later stages, have also been observed. An abnormal dryness of the skin has been noted in severe cases.

It was noted by Vigouroux[72] that in many cases of exophthalmic goitre there was a decided decrease in the normal resistance offered by the skin to the passage of the galvanic current. This he thought was due to hyperidrosis. As this symptom does not occur in other diseases where the perspiration is abnormal, and may even occur in health, it cannot be considered to be pathognomonic.

The hair.—Loss of hair frequently occurs from the scalp, axillæ, and pubes. Thus, Mannheim[73] observed this symptom in fifteen out of forty-one cases, and Yeo[74] reports an interesting case where the hair fell out of the eyebrows and eyelids coincidently with the protrusion of the eyeballs. It is of interest to note that the loss of hair is often unilateral.

Respiratory System.—Disorders of respiration are not uncommon and are occasioned either by irritation or pressure. Among the former ma[illegible]led the attacks of coughing which resemble laryngeal crises.

In many of these cases the dyspnœa may be very great and death from asphyxia may ensue. There may be but little secretion, but at times there is a profuse bronchorea, which is conceded to be due to vasomotor paralysis.

Digestive System.—Disturbance in the gastro-intestinal tract occurs in at least half of the cases, and usually takes the form of a serous diarrhœa, which appears in paroxysms and without colicky pains. These attacks may be most persistent, and may even end fatally.

Generative System.—Although many observers are of the opinion that menstrual disorders are a frequent cause of exophthalmic goitre, there is no evidence to prove this or to indicate that the quite frequent disturbances in menstruation which occur in conjunction with this disease are more than functional derangement, the result of anæmia and chlorosis.

Urinary System.—Polyuria is of common occurrence, and is dependent upon vasomotor activity, being analogous to the excessive sweating and the diarrhœa. Transitory albuminuria has been reported in a number of cases. Glycosuria is a frequent symptom, depending in some cases upon true diabetes, in others upon simple so-called alimentary glycosuria.

Anæmia, chlorosis, and any condition which lowers the vitality, such as prolonged illness, loss of blood, amenorrhœa, sexual excess, the abuse of alcohol, and syphilis, may predispose to the development of the disease; cases have been reported also where diseases of the nose and pelvic organs were thought to have originated the symptoms. Among the exciting causes are sudden fright, great grief, prolonged fatigue, and violent physical strain.

COURSE, PROGNOSIS, AND FREQUENCY.

The disease begins, as a rule, very suddenly, symptoms of palpitation and tachycardia rapidly developing after a physical shock. These either persist, or, what is more common, subside, to be re-excited again by similar causes until the symptoms remain permanent. The future course of the disease is usually a chronic one, and it may persist for years with occasional variations in severity. Complete recovery occurs in some cases, but, as a rule, some of the symptoms remain until death ensues from some intercurrent disease.

Prognosis.—The prognosis, as far as life is concerned and also for a comparatively short duration of the disease, is good, provided the cases are submitted early in their course for treatment and that the subjects are young. The prognosis is more favorable in women than in men. Though rapid onset may be followed by rapid recovery, recovery is usually more rapid and permanent in cases where the symptoms develop more slowly. The appearance of grave complications, such as vomiting, diarrhœa, and the signs of cardiac failure, are always of serious import, as they may occasion a fatal termination. Exophthalmic goitre of itself is seldom fatal.

Frequency.—Exophthalmic goitre is a rare disease; it occurs in all lands and shows a preference to affect individuals between 20 and 30 years of age. Thus, of four hundred and ninety-five cases collected by Buschan (four hundred and seven females and eighty-eight males), the statistics were as follows:

	Females.	Males.
From two and a half to ten years of age	12	3
From eleven to fifteen years of age	17	11
From sixteen to twenty years of age	83	10
From twenty-one to thirty years of age	144	19
From thirty-one to forty years of age	79	17
From forty-one to fifty years of age	51	18
From fifty-one to sixty years of age	21	10

Sex.—Buschan collected nine hundred and eighty cases in the literature, and found of this number eight hundred and five to be women and one hundred and seventy-five men = 1:4, 6.

PATHOLOGY.

Since the recognition of Graves's disease as a distinct morbid entity numerous theories have been advanced to account for its causation, and it has variously been imputed to a dyscrasia, to intestinal intoxication, to disease of the nervous system, and, finally, to a disturbance in the action of the thyroid gland itself. Of these theories the most tenable are those which are based upon either a disease of the nervous system or of the thyroid gland. Regarding the former, it may be said that although observers have designated nearly every portion of the nervous system as the primary seat of the disease, our present knowledge of the subject permits us to eliminate all parts other than the medulla oblongata and the sympathetic nervous system.

While it may be stated that it has been demonstrated that symptoms of exophthalmic goitre may be occasioned by interference with the nerve-centres in the medulla oblongata, and though definite lesions have been found in this part of the brain in some autopsies which have been made upon fatal cases of this disease, there is not yet sufficient evidence to prove that an organic lesion of the medulla is necessary to produce exophthalmic goitre [illegible] is there ground to warrant the assertion that the sympathetic r [illegible] m is primarily at fault, though many of the chief symptoms of the disease may be explained by an affection of that part of the nervous system, and though lesions have been found there in cases of Graves's disease.

As regards the gland itself, while it is evident from microscopical studies that there is a great increase in the amount of the secreting gland tissue in exophthalmic goitre, and from other observations that it is not improbable that there is an alteration in the character of the secretion as well as its quantity, and that the symptoms of the disease may be due to an action of an excess of this altered secretion of the gland upon the tissues, and especially upon the nerve nuclei of the medulla, the entire question of the essential factor or factors in the production of exophthalmic goitre must still remain *sub judice.*

TREATMENT.

The treatment of Graves's disease resolves itself into a general hygienic, medical, electrical, and surgical.

General Hygiene.—The chief indication in the treatment of Graves's disease is rest. Most patients do best upon a prolonged course of strictly enforced rest cure, the treatment being protracted over a period of one or more months of absolute rest in bed. Where this is impracticable or seems undesirable, bodily and mental rest, as far as is possible, should be installed, with the avoidance of all but the slightest amount of physical exercise and the withdrawal of the patient from all contact with that which might worry or excite the mind. No specific regimen can be prescribed as suitable for all cases, the physician being compelled to adjust the amount of exercise and rest for each individual. All violent exercise should be proscribed, and gymnastic exercises, except perhaps the passive mechanical exercise employed by Bryson, are to be avoided. Patients do well by the sea, and a bracing air is usually of advantage. High altitudes should be avoided. Long sea-voyages are often beneficial, provided the patient is not subject to sea-sickness. Alcoholic stimulants are only to be prescribed when the heart is very weak or assimilation very poor. Tea, coffee, and tobacco should be prohibited. The free consumption of milk should be encouraged, and simple and readily digested foods selected; but the diet should always be prescribed with reference to the conditions revealed after an analysis has been made of the gastric contents. If massage be administered, it should be given gently and not too frequently. Cold baths are harmful; warm baths and occasional shower-baths at 100° F. are of benefit. The application of Leiter's ice-coil over the thyroid gland and occasionally over the heart will often diminish the exophthalmus, as well as the vascular excitement.

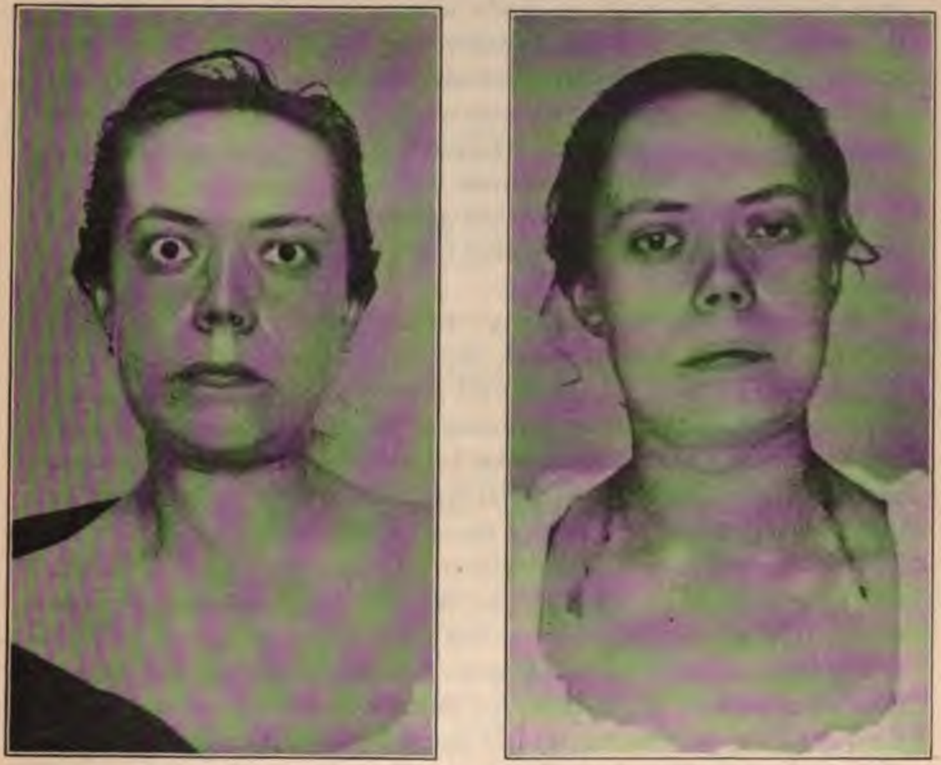
Medicinal Treatment.—The indications to be met in the administration of drugs are to lessen the excessive and abnormal secretion of the thyroid gland, to quiet the general nervousness, to build up the general health and overcome anæmia. A most efficient means to reduce the size of the gland consists in rubbing a small piece of red iodide of mercury ointment over the gland each night until the skin becomes too tender for the patient to bear its further application. After a few days of interruption, this treatment may be resumed and continued with similar interruptions for several weeks or months, or the skin over the gland may be painted with tincture of iodine or anointed with a compound consisting of half a drachm of extract of belladonna and one ounce of iodine ointment.

Belladonna may also be administered internally, beginning with ten minims of the tincture three times daily and continued until secretion has been appreciably affected. Iodine and the iodides have also been of advantage when given by the mouth in some cases, though in others they have been harmful. Bromides are of service in controlling the nervous irritability, and sulphonal, in addition to its action as an hypnotic, has been found to have a favorable effect upon the cardiac and general nervous systems. The valerianates, sumbul, and asafœtida are also of service. Musser relies as standards upon iron, cod-liver oil, and opium. Powdered opium is administered in doses of a quarter to a half of a grain to quiet the erythresium, control the diarrhœa, and stimulate the heart. Strophanthus is often of service, but contrary to what might be expected, drugs that slow the pulse, like digitalis and strophanthus, have but little influence in controlling the disease, though they should be administered when the heart is weak. Ergot is often of decided value, and iron and quinine are useful if there is anæmia.

An extract of the thymus gland has done good in some cases, but the results from its use have been disappointing. Thyroid extract should always be used with caution, on account of its liability to increase the symptoms. Powdered oxide of zinc may be employed to check the sweating and control the diarrhœa.

Electrical Treatment.—Galvanism is of decided value in many cases, and is best applied by placing the cathode over the angle of the jaw, first upon one side and then on the other, while the anode is held upon the back of the neck over the seventh cervical vertebræ. The current should be applied for five minutes at a time, three times daily, in the strength of from two to three milliampères. Vigouroux employs the induced current, the positive electrode being placed upon the neck and the smaller cathode over the carotids; this current should be applied once daily, ten [illegible] minutes at a time.

Surgical Treatment.—On account of the uncertainty attending operative procedures for the relief of exophthalmic goitre and the danger by which they are surrounded, none of the various forms of operations which are about to be mentioned should be tried until all medicinal and other therapeutic methods have failed, and should only be resorted to then when the disease is progressing and the symptoms distressing or dangerous.



A.—The patient before operation. B.—The same patient shortly after.

FIG. 6.—SHOWING THE EFFECT OF BILATERAL RESECTION OF THE CERVICAL SYMPATHETIC. (Jonesco in International Clinics, Vol. I., 13th Series.)

Degeneration of the heart or serious involvement of the nervous system are contraindications, except in the rare cases where the enlargement of the thyroid is so great that the trachea is compressed, when a part of the gland should be at once removed. According to Buschan, the most suitable cases for operation appear to be those in which the enlargement of the thyroid gland has existed for some time previous to the development of the other symptoms.

From the statistics which have been compiled by Kocher and others, it would appear that operative measures hold out a fair chance of improvement or even of cure of cases of exophthalmic goitre, even after all medicinal measures have failed, and that the ligature of the thyroid arteries is equally efficacious and less serious than thyroidectomy.

In 1896 Jonesco resected the superior and middle cervical ganglia for exophthalmic goitre, and the operation has been performed a number of times since by others. Though the number of cases which have been operated on is small, no deaths have occurred thus far, and while the results obtained by this procedure are inferior to those observed after ligature or partial thyroidectomy, the operation is of value and may be used to supplement the action of either of these operations in the event of the persistence of symptoms, such as tachycardia, and exophthalmus, after their performance.

Subjects of Graves's disease bear general anæsthetics badly, so that whatever the operation, it is essential that it should be performed under local anæsthesia.

Treatment of the Exophthalmus.—In the very early stages a light pressure bandage often decreases the exophthalmus, especially where the perfect closure of the lids is still possible; when the exophthalmus is pronounced, tarsorrhaphy, or union of the edges of the lids, is indicated, not only for cosmetic reasons, but also as a prophylactic measure against corneal ulceration. Lateral tarsorrhaphy consists in shortening the palpebral fissure by uniting the edges of the lids at the canthi. This is accomplished by an incision into the margin of the upper and lower lids, which separates the skin from the cartilage, thus permitting the anterior layer containing the cilia to be everted and the groove between the two layers to gap; the raw surfaces of the lids are then approximated by sutures. To insure a good hold on the edge of the lid and to prevent the sutures tearing out by the pressure of the globe, it is advisable to introduce the needles several millimetres beyond the border of the lid, and not including the latter. At times it is necessary to combine a median tarsorrhaphy with the lateral, in which event a narrow strip of skin is excised from the ciliary border of the upper and lower lids at their middle third, and the lids then united in the manner just described.

Treatment of Corneal Ulceration.—Even in cases where corneal ulceration is not present, the precaution should be taken to flush the conjunctival cu[illegible] two or three times daily with a solution of boracic acid (gr. x[illegible] When corneal ulceration is present, greater care should be t[illegible]sing the eye, and two or three drops of a solution of atropine (gr. iv ad f ℥ i) should be instilled night and morning. Hot compresses are of service and should be employed for one-half hour at a time three or four times daily. The ulcers should be carefully cleansed with peroxide of hydrogen (50 per cent.) and a firm compress bandage should be carefully applied. After the inflammation has subsided, if the central part of the cornea is occupied by an opacity, it may be possible to improve vision by an iridectomy. If perforation occurs abscission of the anterior segment of the globe or even enucleation may have to be resorted to.

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CHAPTER XX.

THE PSYCHOLOGICAL EFFECTS OF OPERATIONS UPON THE EYES.

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THIS subject may be studied from two stand-points,—first, from that which deals with the effect upon the mind of an operation which restores sight to individuals who were born blind or who became so by accident or disease, and, second, from that which considers the impression such operations produce upon the mentality in the nature of a shock, quite independent of any influence which might be exerted by the restoration of sight. Before considering either of these phases, it is necessary to inquire whether the mental state of the congenitally blind differs from that of those born with all their faculties, and to what extent loss of sight affects the mind of individuals who have had both mind and sight developed. In answer to these questions it may be stated that in general, blindness in no way affects the mind in its development, and the congenitally blind possess as good a mentality as their fellows. It is a matter of experience also that subjects with healthy minds, who have lost their sight after their mental powers have been developed, suffer no impairment of reason, apart from the general depression consequent upon the realization of how much they have been deprived, normal cerebration being continued without interruption. In individuals, however, who are intellectually weak, it can be readily understood how the loss of so important a sense as sight might be of serious moment, even sufficient, perhaps, to break the links in the fragile chain of cerebration and cause insanity. Notwithstanding this, instances of serious mental lesions from visual defects are very rare and blindness as a causal factor in the production of insanity is mentioned but seldom in alienist literature. It is probable, however, that were a more careful etiological study made of the blind insane from an ophthalmological stand-point, that a connection between the mental and ocular conditions would be traced more often. The cases which have been reported, however, are especially significant, for they demonstrate that not only can insanity be traced to loss of sight, but that the relief of the blindness by operation can restore the mental condition.

RELIEF OF INSANITY BY EYE OPERATIONS.

The literature contains but few cases where it could be definitely ascertained that an improvement in the mental state was noted after such operations, for, although cataracts have been removed from the insane many times, either because the subjects who were operated upon had become chronically insane by reason of the long continuance of the blindness, or because their blindness was of accidental occurrence and in no way responsible for their mental deficiency, the mind was unchanged by the operation in nearly all instances. A few cases, however, where a connection could be traced between loss of sight and loss of mentality have been reported, in which restoration of sight by operation was followed by complete recovery of mind. Thus, the writer would refer to two cases which he reported in 1900, before the County Medical Society of Philadelphia. The first case, which was seen in the wards of the State Hospital for the Insane at Norristown, a male, 45 years old, had been practically blind for 3 years with cataracts. This patient had inherited a disposition towards insanity from his father, who had had acute mania, but his mind had been apparently unaffected until his vision failed, when he became greatly depressed and irritable and developed homicidal and suicidal tendencies. Rest and tonic treatment were prescribed without avail, the mental condition showing no improvement. The connection between the loss of sight and the insanity being so obvious, the cataract was removed by operation and practically normal vision regained. The improvement in the mental condition was immediate and convalescence was so rapid that in a month the patient was discharged from the hospital apparently well. Shortly after, a clerical position was obtained, in which he has continued without relapse from sanity during the past six years.

The second case occurred in a woman, 60 years of age, who had developed melancholia, with failure of sight consequent upon cataracts forming upon both eyes, five years previous to the writer's first observation of her. The mental condition was extremely bad: the patient being excessively violent at times and never entirely rational. Vision was restored by operation and was followed by a rapid and permanent return of a normal state of mind. Cases analogous to these have been reported by Bouissin and Meiser. That of the former author referred to a young man who had lost his mind with the development of cataract and recovered it when the cataract was removed. In Meiser's[1] patient, a woman, 68 years old, also the subject of cataracts, hallucinations similar to those observed after operations upon the eye developed with the failure of her vision but disappeared after the removal of the cataract.

In addition to these cases where the mental disturbance was made to disappear by the restoration of sight, cases have been reported in which the removal of peripheral irritation by enucleation of the globe was followed by the relief of severe mental symptoms. Thus, Moulton reports a case in which progressive melancholia, which had developed secondary to an irido-cyclitis of long standing, was immediately relieved by removal of the eyeball. Of a similar nature was a case recently reported by Hansell.[2] This patient, a female 25 years of age, had been born blind, but during her youth had given evidences of average intelligence and had some school training in a blind asylum, where she had later become a teacher. After some years she developed pain in the head and delusional insanity, the delusion taking the form of the envelopment of all objects in a bright red glare. The eyes were injected, tender to the touch, sightless and atrophic, and as it was thought that they might be the possible source of her delusion, both were enucleated. The patient gradually became more quiet and in a few days after the operation her mind regained its former clearness, and all delusions vanished, so that she was enabled to resume her occupation of teaching. The relief of similar symptoms by operation has been noted by De Schweinitz[3] and Risley.[4]

From these cases it would appear that grave mental disturbances may follow affections of the visual apparatus and that relief may be obtained by the removal of the cause, and although the writer has been told by alienists that many forms of insanity may be temporarily improved if not entirely relieved by any form of operation, or even by shock, or by any force which strongly impresses the mind, he believes that the connection between cause and effect in the cases which were reported by him was too close to deny the direct influence of the restoration of sight upon the mind. He would not deduce from this that the mind can be restored to all blind insane by similar operations, but he is convinced that every effort should be made to give useful vision to all such cases, for although in many instances operations may be unavailing, yet the return of even a few such subjects to society will amply compensate for many failures.

MENTAL DISTURBANCES AFTER EYE OPERATIONS.

This subject refers to the second category contained in the opening paragraph and relates to the impressions made upon the mind by the operation itself, independent of any influence which might be exerted upon it by the restoration of sight. Such impressions develop as a consequence of the shock to which the patient is subjected at the time of the operation and as a result of conditions which arise afterwards, and are of the nature described by neurologists as traumatic neuroses.

In contradistinction to the phase of the subject which has just been considered, where it was shown that restoration of sight rarely exercises any decided action upon the mind, it not rarely happens, after operations upon the eye, that those who are operated upon develop more or less disturbance of mentality. This derangement, as a rule, takes the form of a delirium which appears usually upon the evening of the third day after the operation and continues a few days to a week, according as the surgeon is prompt in combating it, and fortunate in his choice of remedies. Although frequently retarding the healing process by the irritation which is provoked in the eye at the height of the delirium by interference with the dressings, etc., the ultimate success of the operation is not as a rule marred by its occurrence. It is rarely seen in the young, but it is met with in those who are well advanced in years, and, according to the majority of observers, especially in those who are predisposed by heredity, alcoholism, or some other cause, to mental aberration.

During the performance of the operation and for the first few days after it, the behavior of the patients is usually all that can be desired. At the end of the second or third day, however, towards evening, the patients become restless and wish to rise out of bed, and if unrestrained would tear the dressings from the eyes. Rapid and violent delirium may supervene. Such patients usually fancy the room on fire in which they are confined, and would make their escape at all hazards. Not rarely they imagine they are subjects of persecution; that they are being pursued or unjustly confined, and they demand a knife with which to wreak vengeance upon their oppressors. Although the symptoms usually are more apparent at night, the patient rarely recovers his full senses during the day. Hallucinations are not always present, but there is usually some fixed idea which predominates. Pain in the eye is but rarely complained of, and rise of temperature is accidental and not constant. After a few days the delirium subsides and the mental faculties are slowly regained without leaving any permanent mental aberrant processes.

Until Dupuytren,[5] in 1819, differentiated a purely nervous type in non-alcoholics, all forms of delirium occurring after operations were thought to be due to alcohol and were considered to be instances of delirium tremens. This distinguished surgeon saw this nervous form of delirium develop after a number of diverse general operations, though it is of interest to note that in one instance it was recorded that the mental symptoms appeared after the couching of a cataract. In 1863, Sichel[6] contributed the first article which was directed exclusively to the consideration of mental disturbances after operations upon the eye and reported eight cases of delirium following the removal of cataract in old people; later in the same year monographs appeared upon the same subject by Lanne,[7] Magne,[8] and Borelli.[9] In his classic article upon operations, in the first edition of the Graefe-Saemisch Handbuch, Arlt refers briefly to the subject, stating that in addition to delirium tremens, a peculiar delirium appears during the first few days after the operation, chiefly in old persons of an anxious temperament, who are broken down in health.

The next observation of importance was by Schmidt-Rimpler,[10] in 1878, who asserted that mental disturbance could originate not only after operations upon the eye, but could develop even without an operation in individuals whose eyes had been bandaged or who had been confined in dark rooms. In 1890 the entire subject was discussed very thoroughly before several French ophthalmological societies, without any definite conclusion being arrived at, regarding the prophylaxis or etiology of the disturbances, and though a number of communications have appeared since that time, those phases of the subject still remain obscure.

From a comparison of the mental disturbances which occur after operations upon the eye with those which follow other operations upon the body, it appears that both processes are similar, and just as the delirium and mental disturbances arising after operations elsewhere may asume a variety of forms, so the psychoses after operations upon the eye may vary in their manifestations, and while the picture of the delirium which has been given above describes the usual symptomatology, the mental disturbance may assume other forms, being influenced largely by the individuality of the subject affected. Thus, the time of the appearance of the symptoms may vary, and instead of arising within the first few days, the delirium may not manifest itself until the 6th or 7th day and may be retarded even for two or three weeks. In like manner the convalescence may be protracted, and the symptoms instead of disappearing within a few days may persist for weeks or even months. While a mild delirium is the rule, the nervous disturbance may take the form of an active mania and though relapses are very rare and the prognosis is good in most ca[illegible] patient regaining his normal mind at the end of a week, in ot[illegible] the delirium passes into the deepest melancholia, and suicide may result. On account of these variations in the symptoms and course of the disease, Frankl-Hochwart[11] has divided these psychoses into four groups: 1. Hallucinatory mental derangement in non-alcoholics. In this class the commencement of the affection is usually on the first day after the operation and occurs in individuals ranging from 30 to 90 years of age. 2. Simple confusion of ideas in senile individuals. Hallucinations are not present in this class, and dementia not rarely follows. 3. Delirium in alcoholism. In this class the delirium appears earlier and subsides more rapidly. 4. Confusion of ideas due to inanition, occurring in extremely marasmatic individuals and followed by death.

Knapp[12] has made a still more practical division of the cases into three clinical varieties, (*a*) Light cases, exhibiting but slight mental disturbance, and occurring in eccentric and odd characters, (*b*) those exhibiting violent mania, (*c*) persons of a mild melancholic disposition with temporary aggravations and suicidal tendencies.

In attempting to classify cases of mental disturbance after operations upon the eye, Schnabel[13] holds that the delirium is analogous to the acute delirium which is seen in the aged, though its presence in so many young subjects suggests important differences. Mendel[14] regards the disturbance as a form of delirium hallucinatorium, including under this heading, in addition to the mental symptoms after operations upon the eye, other traumatic psychoses as well as senile delirium. He insists upon the necessity of strongly differentiating other types of delirium from senile debility, for he believes that organic changes are rarely absent in senile debility and that the prognosis is consequently bad, whereas the prognosis in other forms, which he considers as being purely functional psychoses, he regards as being the most favorable among mental disorders.

Löwy[15] holds much the same view as Mendel, believing that the mental disturbance is a typical psychosis with a characteristic set of symptoms, and regards senile delirium as a stage of senile dementia. Schmidt-Rimpler[16] classifies the delirium among the paranoia, with the understanding that primary insane conceptions and hallucinations should be regarded as the chief symptom of paranoia.

As might be expected by reason of the greater number of times the operation for cataract is performed upon the eyes of the aged, a class of individuals who are especially liable to be affected by mental symptoms after operations or shock of any kind, the number of times this complication is met with after the removal of the lens, is far greater than after any other operation upon the eye. There are no statistics in the literature regarding the frequency of the disease, with the exception of those drawn from a summary of 24 cases, collected by the writer. Of these 24 cases the delirium appeared 11 times after simple extraction; 8 times after the combined operation; 3 times after iridectomy for glaucoma, and twice in traumatic cases where no operation had been performed upon the eyes or adnexa. Sixteen of these cases were collected from the Wills Eye Hospital records and extended over a period in which 770 cataract extractions had been performed in that institution, making a percentage of $2\frac{6}{77}$ per cent. It was felt by the writer,[17] however, that these figures might be misleading and mental disturbances might be much more common, as the custom which prevails in that hospital of administering hypnotics at the first symptom of mental unrest might have prevented the development of what would have been a true post-operation psychosis. Of 12 cases observed by Kipp, 5 followed cataract operation, 1 an iridectomy for glaucoma, 3 traumatic injuries of the eyeball, and 3 corneal ulceration. Schnabel [18] saw 12 cases of delirium among 183 cases of senile cataract in his own clinic at Innsbruck, though in Jäger's clinic in Vienna delirium occurred but 11 times among 1500 cases of cataract. All of Schnabel's cases occurred in old subjects and several times after iridectomy without removal of the lens. The psychoses began 20 hours to 6 days after the operation and lasted from 1 to 4 days. In half of the cases the mental symptoms occurred at night and in the other half during the day. Convalescence was rapid and without complications. Löwy explains the unusual frequency with which Schnabel saw cases in Innsbruck and its rarity in Vienna, by the fact that the inmates of the hospital in Innsbruck were, for the most part, inhabitants of the mountains and unaccustomed to restraint and confinement and more liable to be affected by such conditions than the city-bred Viennese. He says that the same explanation applies to Christen of Pavia's cases also, the frequency with which delirium occurred in his clinic in that city being accounted for by the unrestrained and rather wild character of the inhabitants of that neighborhood.

As a rule, recovery is more rapid, the symptoms less violent and the prognosis generally better among the aged than the young. Webster [19] saw a fatal case, as the result of meningitis, secondary to panophthalmitis, which resulted from a patient with delirium tearing off the bandage. Berry [20] recorded epileptic attacks following an intraocular hemorrhage complicating cataract extraction.

Etiology.—As would be expected from the nature of the affection and our ignorance regarding the pathogenesis of psychoses in general, many theories have been advanced to explain the origin of the affection. Sichel thought the delirium was due to a change in the disposition of the individual, which resembled nostalgia. A number of authors, however, assume a special tendency towards mental derangement in all who become delirious following operations upon the eye. Among these may be mentioned Arlt,[21] Schmidt-Rimpler,[22] Knies,[23] Warloment,[24] Hirschberg, [25] and Valude.[26] These authors believe that mental symptoms arise in only very weak and nervous people, but fail to mention further characteristics of this predisposition. Lopez thinks that the delirium occurs only in those who are already actually mentally alienated, although usually in a latent form, the mental weakness having passed unperceived by the physician.

Among occasional exciting causes may be mentioned Parinaud's[27] explanation of extreme preoccupation of the patient for several days preceding the operation itself, and the withdrawal of all external impressions during the first few days after the operation, resulting from the absolute rest in bed, and the darkness and seclusion enforced by the closed eyes. Schmidt-Rimpler[28] is strongly of the impression that one of the chief factors in the production of the delirium is the bandaging of both eyes, and in support of this theory cites two cases in which no operation was performed, but who developed delirium, as he thought, simply by reason of their being confined in a darkened room during the treatment for their ocular affection. One of these cases was that of a young man, 19 years old, who was suffering from iridochorioiditis; the other that of a man, 59 years old, who had plastic iritis. This theory is supported by Sichel,[29] Swanzy,[30] Valude,[31] Borelli,[32] and Armaignac;[33] the last three authors giving the notes of cases where the delirium disappeared with the removal of the bandage.

Another set of authors, including Galezowski,[34] Salvator, Angela Ledda, and Lemkiewicz, attributes the delirium to the action of atropine. Grandclement[35] subscribes to this theory also, as the mental condition of a case observed by him was made worse after each instillation of this drug.

As just mentioned, Schnabel was of the opinion that the psychosis was merely a form of senile delirium and he explained the transitory nature of the disturbance in mentality as well as its sudden appearance, to circulatory changes in an atrophic brain. He thought that the hyperæmia of the brain is favored by the prolonged rest upon the back, with the stillness and darkness, to which the patients are subjected while in a more or less excited condition. He does not believe Schmidt-Rimpler's theory of the exclusion of light, as the sole cause, as he had observed the condition to arise after the bandage had been removed, and calls attention to the rarity with which delirium occurs in the large number of young persons who are treated in darkened rooms and with bandaged eyes. Kraft-Ebbing[36] is of the opinion that fear is the causal factor, and thinks that the symptoms rarely develop in those who have normal minds, but rather in those who are abnormally excitable, in imbeciles, or in those weakened by senility, alcohol, or sickness. Frankl-Hochwart called attention to the connection between affections of the nerves (especially neuralgia) and psychoses and also to the fact that mere injury of the globe, as in the cases reported by Griesinger,[37] Arlt, and Fürstner, could occasion delirium and even death, and ascribes the delirium to an affection of the sensory nerves of the eye conjoined with senility, darkness, absolute rest in bed and the isolation from the external world.

An additional theory is mentioned by Berger,[38] who says that he remembers that Meynert, of Vienna, ascribed the production of the delirium to the excision of part of the iris, which is one of the steps in the common operation for cataract. Berger, however, remarks that this theory is no longer tenable, since Parinaud has observed the delirium following the extraction of cataract without iridectomy.

The restriction in diet and lessening of the amount of food immediately before and after the operation has also been advanced as a cause. Dupuytren was the first advocate of this theory, as he found that after he gave his patients sufficient nourishment, mental derangements occurred much more rarely. Parinaud and Gillett de Grandement also support it, for both think that the weakness induced by the restricted diet is an important factor in precipitating the delirium; the latter author citing a remarkable instance which was evidently induced by this cause.

While it is evident that in alcoholism the excitement consequent upon the operation, conjoined with the depressing circumstances which are attendant upon the after treatment, may easily provoke a delirium of persecution, it is not the purpose of the writer to consider this type of cases in this paper. In this connection, however, reference may be made to a case reported by Lopez,[39] where the delirium occurred in an alcoholic, in whom the eyes were not bandaged and where the wound healed perfectly. Lopez thinks that it is a circumstance worthy of consideration that he has always observed the delirium in the old and atheromatous, and says this has led him to think that this disease of the vascular system plays an important rôle in the production of cerebral troubles, and subsequently of maniacal paroxysms. He adds that it is not strange that the cause of the delirium in question should be charged by some to alcoholism, as this is a frequent factor in the production of atheromatous degeneration.

Martin [40] also, who has contributed a recent and comprehensive article on the subject, holds that the delirium is due to inanition, but thinks that this is induced chiefly by the withdrawal of alcohol. He does not believe that the mental derangement in this class of cases is an alcoholic delirium in the sense that the subjects are ever intoxicated by alcohol, but that it is delirium occasioned by the privation of this agent in individuals who are accustomed to it in moderate amounts; for he thinks that the brain, especially in the old, which has been habituated to act normally under the influence of alcoholic stimulation, becomes deranged if this agent is withdrawn.

Fernandez [41] believes the abstinence from customary alcoholic stimulation is the determining factor in producing the delirium. He has observed the conditions six times in a very large experience, and only after cataract operations.

In a paper which was presented at the Congress of Alienists of La Rochelle, in 1893, Regis and Chevalier-Lavaure considered the delirium which is observed after operations in general to be due to autoinfection as a result of uræmia and toxins originating in the intestines, and thought that the mental disturbance in these cases was analogous to those occurring in typhoid fever, influenza, and erysipelas. Fromaget subscribed to this theory as being operative in most cases of delirium after operations upon the eyes and was of the opinion that the conditions favoring autoinfection were found in the disturbances of the liver, kidney, and heart, which are common to the aged. In support of his views, he gave the notes of a man aged 92 years, in whom an anuria prevailed two days after the removal of the lens, the flow of urine being increased after some difficulty by injections of caffein. This theory of autointoxication has also been subscribed to by Finlay [42] from conclusions based upon the study of a case where there were signs of uræmia.

Kipp [43] in a recent article, in which he reported 12 cases of delirium after operation and injury of the eye, concluded from a study of these cases that the "psychoses were the result of a change in the patient's environment and to an increasing longing to get away from the new surroundings." He refers to the condition as a homesickness, or nostalgia, which may end in a form of melancholia with homicidal and suicidal propensities.

Such are the theories which have been advanced to account for this strange form of mental disturbance, and while in the present state of uncertainty regarding the nature of psychological processes in general, it may be impossible to determine the precise etiological factor which occasions the delirium, there are such decided objections which can be offered to some of them that they may be eliminated from the list. Thus, the assertion that the mental aberration is due to a predisposition to mental unsoundness can be disproved by the absence of symptoms indicating insanity in the previous history of most of the patients, and by the transient character of the delirium. In the few cases in which the mind does not recover its normal condition after one or two weeks, but lapses into mania and melancholia, it is fair to suppose that some such conditions may have existed previous to the operation.

Martin could satisfy himself of a state of mental weakness prior to the operation in but two cases mentioned in the literature,—*i.e.,* that of Warloment, where the statement is made that his case seemed mentally "innocent," but that he had never given another sign of delirium or insanity; and that of Hirschberg's, where the patient was said to have some mental unsoundness. He says that in none of the 20 or more other cases mentioned is there any reason to suspect a prior predisposition towards insanity. Lopez's claims of a mental predisposition should not be regarded, as two of his cases were probably quite insane before the operation, and should not really have been described as instances of post-operative psychoses; one of the cases occurring in a family of eccentric members and the patient remaining insane for two months after the operation, while the second case was melancholic.

Sweet's[44] case belongs to the same category. This author cites an instance of delirium of persecution following a cataract extraction which was evidently excited by the fear of going blind. The patient was very nervous, and although there was no former history of insanity, there was probably a strong disposition toward mental weakness. The patient died two months after the operation.

Of the 24 cases reported by the writer, there were but 3 of questionable healthy mentality.

In opposition to the bandage theory is the fact that even after it is removed from the unoperated eye, there is usually no improvement in the mental condition. Thus, neither in Warloment's and Hirschberg's cases nor in any of those cited by the writer was there any improvement after the bandages were removed. Gorecki, also, and Calderon[45] saw delirium after cataract operation in non-alcoholics, when the eyes were not bandaged, and the same was true in a case observed by the author.

This would seem to be in exact contradiction to a case reported by Valude, for, in this instance, as soon as the bandage was removed from a 75-year-old patient, who had developed delirium as a result of double iridectomy for glaucoma, the delirium ceased; and likewise a case of Borelli, where amelioration was almost immediate when coquilles were substituted for the bandage. Martin, however, disposes of both of these objections, or rather exceptions, as follows: Valude's case was a simple coincidence,—*i.e.,* the bandage being removed about the time that the other remedies which had been employed became efficacious, while Borelli's case was not one of true delirium, but simply one of a fixed idea which existed before the operation.

It can also be argued against the bandage and exclusion of light theory, that if the delirium be due to this cause, insanity should be much more common among the blind than is actually the case.

The statistics of the summary, compiled by the writer in his series of cases, show conclusively that atropine, also, could not be considered as a causal factor in the production of the delirium. Cocain can also be eliminated. Indeed it would seem to be proven beyond question that the condition of the eye itself has nothing to do with the development of the mental alienation.

Sichel's and Kipp's theory that the delirium was due to homesickness cannot be true in all cases, for the delirium has been known to originate in many cases when the patient was operated upon in his own home. Many of Kipp's cases seemed to be alcoholic, and the improvement noted in their condition after they left the hospital may have been due to the stimulation of alcohol which they then had an opportunity of acquiring; or, the restraint of the hospital being removed, to the stimulus given their mind by the necessity of self-direction and responsibility.

But three theories remain—that of excessive preoccupation upon the part of the patient prior to the operation, that of the withdrawal of food, and that of autointoxication. Of these, the latter two may be readily disposed of, at least in so far as the cases which came under the observation of the writer are concerned, for he believes that there can be no question of starvation in any of the cases which he reported, as it is his custom to make no change in the diet until 24 hours before operating. At this time the first preparations are made for the operation; the patient is bathed and the eye which is to be operated upon is protected by an antiseptic dressing after thorough flushing with a boracic-acid lotion. A light supper is then administered, which is followed by a laxative pill at bedtime. The following morning the patient partakes of a simple breakfast, and as the usual hour of operation is in the afternoon, a glass of milk and a cracker are given at luncheon. After the operation, milk is again repeated at frequent intervals. The diet of the second 24 hours also is largely a milk one, though broths or beef-tea are given twice to prevent extreme constipation. On the third day the patient is usually allowed to get out of bed and is placed on a soft diet, and if the bowels have not been opened, a laxative pill is administered. On the fourth day liberal and supportive meals are prescribed.

Similarly, he does not think that the recent theory of autointoxication which has been advanced by Fromaget could have operated in any of the cases which he has cited, for care is always exercised to avoid any such complications. Thus it is a rule that the urine of every cataract patient shall be examined the morning of the day of the operation, and care is exercised to avoid retention after operation. The bowels, also, if nature has not already accomplished this, are invariably opened on the third day by a cathartic.

After a critical study of the entire subject the writer has finally arrived at the opinion that while any of the above factors, or others, such as the constraint of the supine position and the unusual stillness of the surroundings, may operate in certain cases as etiological factors, the chief cause of the delirium after operation upon the eyes is largely psychic, and agrees with Parinaud that it is due to the preoccupation upon the part of the patients prior to and after the operation. While they may appear calm and collected and submit to the operation most cheerfully, if one scrutinizes them closely, in most instances he will find that this state of quietude is assumed and that in reality there is great though suppressed excitement.

The very thought of sight after the weeks and months, nay, unfortunately, in many instances, after the years of blindness, is enough to upset their mental equilibrium, and the desire to assist the operator during the operation, and not to wince at pain or to do anything that might mar its success, must also be a great tax on their courage, and calls forth all of their power of self-control; and then, following the removal of the lens, the glimpse, though momentary, which is obtained of the outside world, which has been so long hidden from their gaze, and the realization that the suspense is over, must undoubtedly occasion more or less psychical disturbance.

Treatment consists in improving the state of the cerebral circulation by nitroglycerin and strychnine, and in controlling the delirium by hypnotics, in a supplementary diet, and in the administration of alcohol if the patient has been habituated to the use of intoxicants. As the statistics do not show any improvement in the symptoms to follow the removal of the bandage from the unoperated eye, this step is not to be advised, unless the wound caused by the operation be thoroughly healed. There is also no reason to discontinue the employment of atropine.

Constant oversight and judicious and tactful nursing are most essential, and amelioration in the mental condition frequently follows the installation of a proper person by the bedside.

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CHAPTER XXI.

THE SURGICAL TREATMENT OF INTRACRANIAL LESIONS CAUSING DISTURBANCES OF VISION.

BY CHARLES H. FRAZIER, M.D.

LESIONS OF THE CRANIAL SINUSES AS AFFECTING THE CONTENTS OF THE ORBIT.

Anatomical Considerations.—The venous circulation at the base of the brain through the medium of the cranial sinuses corresponds in one particular especially with the arterial circulation of the same region, in that there is a free anastomosis not only between the sinuses on the same side, but also between the sinuses of the opposite side of the brain. Therefore, a thrombus forming in one sinus may extend by continuity to another on the same or the opposite side. The tissues of the orbit are not disturbed unless there is a lesion of the cavernous sinus; therefore, it is the conditions that affect this sinus that concern us especially.

The cavernous sinuses lie on either side upon the body of the sphenoid bone, and are in direct communication with one another through the circular or intercavernous sinus. Into the anterior portion of the cavernous sinus empty the ophthalmic veins, while the posterior portion through the medium of the inferior and superior petrosal sinuses is in communication with the lateral or transverse sinus. In addition to these the cavernous sinus has other important anastomotic relations, which bring the sinus indirectly into relation with extracranial structures. Of the most importance is the so-called vein of Trolard which, arising in the pterygoid plexus of veins, passes, with the third division of the fifth nerve, through the foramen ovale and empties into the inferior petrosal sinus. These means of communication explain the relation of cause and effect between inflammatory lesions in the distribution of the pterygoid plexus of veins (the alveoli of the inferior maxillary bone, the tonsil, pharynx, etc.) and thrombosis of the cavernous sinus. By way of the temperosphenoidal sinus the infectious agents from the skull may be carried to the cavernous sinus. Not to be forgotten is the anastomosis between the terminal capillaries of the facial and ophthalmic veins. Thus inflammatory lesions in the distribution of the facial vein by a retrograde process may give rise to sinus thrombosis.

Finally the intimate relation between the cavernous sinus and the third, the fourth, the first division of the fifth, and the sixth cranial nerves is not to be forgotten. The pressure of a swollen sinus or of an extrasinous exudate will account for many of the symptoms of thrombosis. As the sinuses have no valves, the blood may flow in either direction. To the absence of valves, as well as to the freedom with which sinuses on one side anastomose with those on the other, must be attributed the non-appearance of obstructive symptoms in thrombosis of some of the sinuses and the difficulties, therefore, attending the diagnosis.

THROMBOSIS OF THE CAVERNOUS SINUS.

Etiology.—The structure of the sinuses is said to favor venous stasis.[1] The triangular shape of the canals increases the frictional resistance, the walls constructed of fibrous tissue are rigid, and the dense bands of fibrous tissue which pass from one wall to the other prevent the collapse of the sinus and in themselves offer resistance to the blood current. Furthermore, the horizontal decubitus naturally predisposes towards venous stasis, so that, in cases of long-standing illness associated with depression of the cardiac action, the effect of this position in co-operation with the diminished force of the blood stream is an important etiologic factor.

In general terms, thrombosis may be due to (1) systemic disturbances, (2) to traumatism, and (3) to a septic process. Systemic disturbances are observed in the very young and the very old. In the first four years of life one observes the marasmic thrombosis of the old writers, as in patients suffering from grave forms of gastro-enteritis, cholera infantum, bronchopneumonia, more rarely tuberculosis and hereditary syphilis, as well as in convalescents from the acute infectious fevers, such as scarlet fever, typhoid fever, diphtheria, and influenza. Marasmic thrombosis almost invariably involves the longitudinal sinus, and the treatment is purely medical. Both glycosuria and albuminuria are believed to exert a predisposing influence in the origin of thrombosis in people of advanced years, but it is more than likely that this relation, with glycosuria especially, may be accounted for by the fact that not uncommonly carbuncles develop in diabetic individuals.

Thrombosis as a result of traumatism is uncommon. The sinus has been injured frequently in operations upon the Gasserian ganglion, but, so far as I know, these injuries have not given rise to any signs of interference with the circulation of the orbit. In the absence of infection, gunshot wounds are more likely to determine an arteriovenous aneurism than thrombosis of the sinus. Fracture of the base of the skull, blows upon the head, and punctured wounds through the orbit, occasionally cause thrombosis. But four cases have been reported in which the sinus has been injured directly without involving the skull.

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[1] Marfan, Traité Maladie de l'Enfance, tome iv.

Infective thrombosis is by far the most common. The source of infection may be intracranial or extracranial, and the thrombus may be either primary or secondary. The intracranial causes include basal meningitis, osteitis of the sphenoid bone, and abscess together with thrombosis of the lateral or other sinuses. According to Germain,[2] secondary thrombosis—*i.e.*, extension from other sinuses—is probably the most common if we accept all cases. Out of one hundred and forty-nine cases in which an autopsy was made, in forty-three the disease originated in the ear, causing thrombosis of all or most of the basal sinuses, and it is fair to assume that the lateral sinus was the one first affected.

The extracranial sources of infection are as follows: (*a*) affections of the orbit, panophthalmitis, chalazion, blepharitis, and conjunctivitis; (*b*) affections of the nasal cavities,—*e.g.*, tubercular and syphilitic ulcerations; (*c*) infection of maxillary, ethmoidal, sphenoidal, and frontal sinuses; (*d*) affections of the mouth and pharnyx, including the tonsils,—*e.g.*, tonsillitis, angina, diphtheria, and alveolar abscess; (*e*) inflammatory lesions in the distribution of the facial vein, furuncle, cellulitis, etc.; (*f*) inflammatory lesions of the scalp. Of one hundred and eighty-two cases collected by Dwight and Germain the source is given as follows: From the eye, 4; nose, 9; mouth, 14; face, 34, and lips, 2.

Pathological Anatomy.—The thrombi of an infective thrombophlebitis are at first red and friable, but later become grayish or yellowish and of firmer consistency. The lumen of the sinus may be completely or incompletely obliterated and the clots are more or less adherent to the sinus wall. The latter becomes thickened and its internal coat becomes lustreless and rugose. Infective thrombi should be distinguished easily from the cadaveric thrombi, which are blue, friable, and not adherent to the sinus walls.

Meningeal or cerebral lesions are more or less widely distributed, according to the extent of the thrombus. If limited to the cavernous sinus, a localized meningitis may develop, which may become diffuse or may resolve itself into an abscess. On the other hand, if the thrombus is extensive and involves a number of sinuses, hemorrhages, usually meningeal, intra-arachnoid or subarachnoid congestion, œdema of the brain, and intraventricular hydrops may be looked for. Large cerebral hemorrhages are seldom seen, and softening of the brain when it occurs is due to the local stasis or the anæmia which the hemorrhage may bring about.

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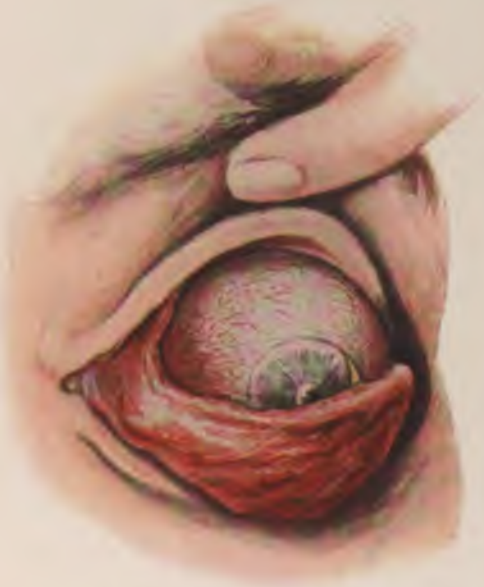
[2] Boston Medical and Surgical Journal, vol. cxlvi., No. 18.

Symptoms.—The symptoms of thrombosis of the cavernous sinus for the most part are due to obstruction of the ophthalmic veins. They are due also to pressure of the sinus upon the adjacent structures, to meningeal irritation, and to general systemic infection. Usually within a few days of the onset of the disturbance of the sinus, although in some cases the interval is much longer, signs of obstruction make themselves apparent; œdema of the eyelid and conjunctivæ and face, haziness of the cornea, choked disk, dilatation and tortuosity of the retinal veins, with œdema of the papillæ and retinal hemorrhages, exophthalmos, due to the infiltration of the cellular tissue of the orbit, ptosis, which may be the result either of the œdema of the upper lid or of paralysis of the oculomotor nerve, dilatation of the pupil; the movements of the bulb are restricted (Plate VII). Pressure of the swollen sinus upon the adjacent structures will excite pain in the distribution of the ophthalmic division of the trifacial nerve and paralysis of the third, fourth, and sixth nerves. While in some cases but one of the cavernous sinuses is involved, in most instances, on account of the free communication between the right and left sinuses, the thrombus extends rapidly, usually within forty-eight hours, from one to the other, and the same clinical picture is reproduced. It has been observed that the development of the symptoms on the opposite side is associated with an equally rapid subsidence of the symptoms in the side primarily affected.

The cerebral symptoms are constant and form a very conspicuous part of the clinical picture: headache which is often most intense and of early onset; cerebral irritation, which may be due to meningitis or to circulatory disturbance and is manifested by the irritable and apprehensive state of the patient; vomiting, delirium, exaggerated reflexes, somnolence and even coma preceding death. These symptoms may be due to the typhoidal state or to the circulatory changes, but when the process has extended to the meninges, typical symptoms of meningitis, such as convulsions and retraction of head, may be present. This latter complication is rare, however, since when the process extends it is more likely to extend in the direction of least resistance,—namely, from sinus to sinus.

To complete the clinical picture should be added the symptoms which are the result of the general systemic infection, fever, chills, rapid pulse and respirations, and so on. As one would expect, pyæmia is a very

PLATE VII.



Original. Showing protrusion of the eyeball, œdema of the conjunctiva, and injection of the corneal vessels attending obstruction of the cavernous sinus.

common termination, and the case becomes further complicated by metastatic deposits in the lung and other organs.

Diagnosis.—It should be borne in mind that obstruction of the circulation in the cavernous sinus and ophthalmic veins is not necessarily due to thrombophlebitis. The pressure of a tumor in the orbit or sella turcica or aneurisms of the carotid artery may so interfere with the venous circulation as to simulate thrombosis. Cellulitis of the orbit and phlebitis of the ophthalmic veins may produce the same effect. In the case of tumors the diagnosis is usually based upon the gradual onset of the symptoms, upon the absence of constitutional signs of infection, and upon the absence of cerebral phenomena. In the absence of cerebral phenomena it would be almost impossible to distinguish between a true sinus thrombosis and a phlebitis of the ophthalmic veins or orbital cellulitis. In the latter case the changes in the fundus and other signs of obstruction would not be so marked, the condition would not spread to the opposite side, and headache would be less intense and more superficial. To differentiate a thrombosis of the cavernous sinus from that of the longitudinal or transverse sinus it should be borne in mind that thrombosis of the longitudinal sinus is attended with cyanosis of the face, with dilatation of veins leading from the great fontanelle to the temporal and facial veins, and with epistaxis. Thrombosis of the transverse or lateral sinus would be preceded in almost every instance by infection of the auditory canal or mastoid cells, and would be associated with œdema of the mastoid region, cellulitis of the neck, phlebitis of internal jugular vein, and perhaps paralysis of the facial and acoustic nerves.

Surgical Aspects.—When one considers that barely seven in every hundred cases of infective thrombosis of the cavernous sinus recover, it would seem that surgeons would have endeavored to enter the field long ago with the hope of reducing the mortality, and yet until recent years no attempt has been made. The treatment of the condition has been said to be preventive only, and no further consideration has been given the matter. The difficulties that arise once we consider seriously the surgical aspects and the surgical possibilities are manifest. In the first place, the condition itself is one which is not easy of diagnosis, and in many cases the diagnosis has not been made at all, or not until metastasis has occurred, or the toxæmia is so profound and the thrombosis so extensive that the case is regarded as desperate, and all hope of recovery is abandoned. Then, again, the cavernous sinus was supposed to be so inaccessible that, even were operative intervention considered justifiable, no effort would be made to reach it.

The principles to be observed in the treatment of this condition are recognized fully and have been applied frequently for the relief of thrombosis of the lateral sinus, and with a great measure of success. If the cavernous sinus could be approached, opened, and drained with as much ease and safety as the lateral sinus, the results of such intervention should be as good in one as in the other. It remained for Dwight,[3] in an operation which he performed in January, 1900, to demonstrate the feasibility of opening and draining the cavernous sinus in case of thrombosis. The case is one of sufficient moment to warrant a brief abstract of it. The patient, a man forty years of age, was admitted to the Boston City Hospital suffering from a small abscess on the right side of his face just above the upper lip. He had in addition all the symptoms of obstruction to both ophthalmic veins, more marked in the right than in the left side. He was in a state of profound toxæmia. Under ether anæsthesia the cavernous sinus was approached by the route recommended by Krause for resection of the Gasserian ganglion. The dura was opened; the tip of the temporal lobe was lifted upward and backward until the cavernous sinus was felt. With a protected bistoury the sinus was opened, and a quantity of dark semiclotted blood gushed out. A strip of gauze was introduced for drainage and the wound closed. The results were almost immediate; within half an hour the exophthalmus almost entirely disappeared, and to a great extent the chemosis and œdema. His general condition improved somewhat, but he died six and a half hours after the operation. The lesson to be learned from this operation is twofold: first, that the cavernous sinus is not without the realm of the surgeon's knife, and, second, that an incision into the sinus on one side will suffice to drain the sinus on the other side. The fact that both sinuses have been involved is not necessarily a contraindication to operative intervention.

While the first recorded operation aimed directly at the cavernous sinus, there have been cases reported in which recovery has ensued after indirect drainage,—that is to say, when the cavernous sinus has become the seat of a thrombus which has extended from the lateral sinus, the latter has been opened and curetted with the intention of draining all the basal sinuses, and in three instances the operation was successful. In discussing the technique of this operation three points are to be considered,—the method of approach, the incision in the dura, and the method of drainage.

The sinus will be found most accessible if approached by the temporal route: the opening in the skull may correspond precisely with the one which would be made for exposure of the Gasserian ganglion. The difference between the two operations would consist essentially in this,—namely, that in the exposure of the ganglion the operation is extradural, whereas in the exposure of the sinus it is almost imperative to make a dural incision and approach the sinus in the space between the base of the brain and the dura. The incision in the dura should be made on a level with the base of the skull, the base of the brain elevated, and, once the sinus is reached, it should be laid open and the thrombus thoroughly removed with a curette. It is most important that the seat of the thrombus should be effectually drained, and to this end a small cigarette drain should be introduced.

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[3] Boston Medical and Surgical Journal, vol. xlvi, No. 18.

TUMORS OF THE OCCIPITAL LOBE.

Tumors in this region may be considered very properly in the category of intracranial lesions giving rise to visual disturbances. If the tumor involves the occipital lobe, hemianopsia will be the only focal symptom; although, if the visual tract be involved on its way to the left occipital lobe, there may be sensory aphasia. The exposure of tumors in this region may be of exceptional difficulty if the tumor is not situated on the surface of the brain, immediately beneath the cranial vault. Tumors situated on the mesial surface or upon the surface in relation with the tentorium are very much more difficult of access. There seems to be a very much greater tendency for the brain to bulge in operations upon the occipital than upon any other lobe, and this of itself constitutes a very troublesome and serious obstacle. In the first place, it makes it harder to retract the brain sufficiently to explore the mesial and tentorial surfaces, and, in the second place, it may prevent proper coaptation of the edges of the dural wound.

The osteoplastic flap for the exposure of this region is quadrangular in shape; the anterior margin corresponds to the line of the occipito-parietal suture, the posterior margin should come as near as one can go with safety to the lateral sinus, the superior margin should be parallel to and not more than one-half inch from the median line, and the base of the flap connects the inferior extremities of the two sides. The time required to reflect a flap in the occipital region will be longer than that for any other region, because the occipital bone is considerably thicker than either the parietal, temporal, or frontal bone. Once the flap is reflected, the surface of the brain thus exposed to view should be carefully examined, and, if the results are negative, a suitable retractor is introduced, first on the mesial, then on the tentorial surface, and a careful exploration, both visual and digital, is made. Because of the tendency of the brain to bulge in this region, once the dura has been reflected, the manipulations necessary for exploration should be conducted as rapidly and as delicately as possible. Rough handling and frequent sponging will rapidly excite such œdema and congestion of the brain tissue as to cause an increase in volume of the affected tissue and greater or less protrusion of brain substance through the dural opening.

Chronic hydrocephalus sometimes simulates brain tumor, and, if the posterior horn of the lateral ventricle be very much dilated, the overlying layer of brain tissue may be so thin as to rupture during the operation, and thus establish a direct communication with the ventricle. In a case under the author's observation this condition was found; at first it was mistaken for a cyst, but further investigation showed that the cavity which had been opened, was the posterior horn of the dilated ventricle.

If, when the exploration has been completed and the tumor (if found) removed, there be any difficulty in closing the dural wound, because of undue protrusion of the brain, it will be necessary to fashion a flap from the pericranium sufficiently large to close the defect left by the insufficient size of the dural flap. The pericranial flap may be dissected from a portion of the skull adjacent to the field of operation or from the segment of bone in the osteoplastic flap. When the tumor has not been found, or if found cannot be removed, it is better to remove the pericranium from the bone in the flap, since for the permanent relief of pressure the osseous flap should not be replaced. The wound in the scalp should be closed throughout with interrupted silkworm-gut sutures and drainage material, preferably rubber tissue, introduced through one or two openings made by incising the scalp obliquely one or two centimetres to the outer side of the margin of the flap. The drainage material may be removed in twenty-four hours and the sutures one day later.

PRESERVATION OF VISION IN CASES OF BRAIN TUMOR.

The most common cause of disturbance of vision in cases of brain tumor is an optic neuritis, leading to choked disk and eventually to optic atrophy. While optic neuritis may be due to other intracranial diseases as well as to extracranial lesions—as albuminuria and chlorosis—the most frequent source is brain tumor, and, according to Gowers, a considerable degree of neuritis is rarely due to any other cause. Optic neuritis is not, as was hitherto supposed, due to increased intracranial tension. In fact, it is rarely due to any single factor, but the most potent factor is the descent of a process of tissue irritation, which when it reaches the papilla, sets up a more intense inflammation. But whatever the cause, once the neuritis begins it will lead eventually to choked disk and finally to optic atrophy and total and irremediable blindness. It is important to bear in mind that this process does not develop always with the same degree of rapidity, and, as a rule, the more rapidly it develops the more intense it becomes. In some cases months may elapse and the neuritis still be of a mild grade, while in others again it may reach a stage of great intensity within a fortnight. The essential points to be remembered with relation to the development of an optic neuritis due to brain tumors are these: first, that, if it is not relieved before the optic nerve becomes atrophied, permanent blindness will be inevitable; and, second, that in its early stages, by the performance of an operation easy of execution and free from danger, the neuritis will subside and vision be restored. In addition to disturbance of vision, patients with brain tumors also suffer from headache and vomiting, and not only the former but the latter as well may be relieved by the same operation. The operation which should be performed in these cases is the *trépanation decompressive* of the French, and consists essentially in the removal of a not inconsiderable section of bone from the cranium. It matters not from what portion of the cranium the bone is removed in so far as concerns the effectiveness of the procedure, although preference should be given to the parietal or temporal region rather than the occipital or frontal. The frontal region is objectionable for cosmetic reasons, and the occipital region because here there seems to be a

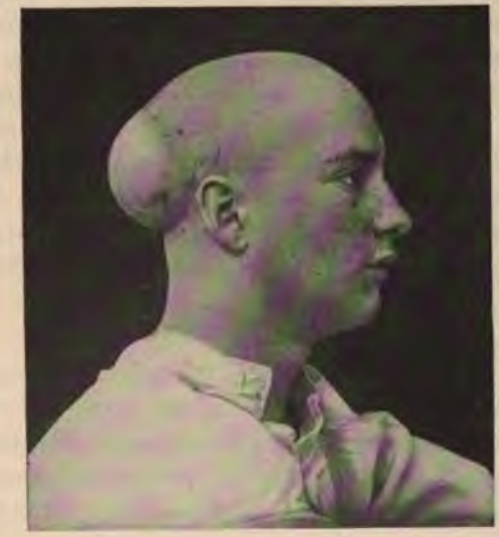

FIG. 2.—Original. Hernia cerebri, showing the very marked protrusion which follows the removal of a considerable portion of the occipital bone in palliative operations for the relief of increased intracranial tension.

tendency to the more rapid development of a hernia cerebri, and in this position the protrusion would be conspicuous and unsightly (Fig. 2). A section of bone six to eight centimetres in its various dimensions should be removed from the parietal or temporal region, first on one side, and later on the other, if after the first operation there are any signs of recurrence. It is not only unnecessary but inadvisable to remove the dura, because of the possibility of a fungus cerebri developing should the wound become infected. The dura may, however, be cut in a stellate fashion, as recommended by Kocher, beginning in the centre of the opening and making several radiating incisions towards the periphery. When vision is beginning to fail, delay or postponement of the operation for any length of time is absolutely unwarrantable. In some cases it may be impossible at first to localize the tumor accurately, but this should not constitute a contraindication, and the surgeon should proceed to operate, if only to save the patient's vision. Needless to say, the indications for operation are quite as positive in inoperable growths. There is no treatment, whether medicinal or surgical, which gives a greater measure of relief to patients suffering from an incurable lesion than the so-called *trépanation decompressive.* The relief from headache and vomiting is often immediate and permanent, and if the optic neuritis is not of too long duration the patient's sight will be restored.

TUMORS OF THE CHIASM.

In the great majority of instances the optic chiasm is injured by tumors taking their origin from the pituitary body, and of these a considerable proportion occur in cases of acromegaly. No attempt has been made to expose or remove these growths, because they are believed to be inaccessible. Were it not for their inaccessibility many of the tumors could be removed, because a large proportion of them are benign. The malignant tumors, probably under any circumstances, should be regarded as inoperable. Owing to the firm attachments of the dura to the various processes at the base of the skull, an extradural operation would be impossible. Even though, by the temporal route, one should succeed in reaching the foramen lacerum anterior, which in itself is not so easy of access, the sella turcica and the processus clynoideus posterior would still have to be passed. Kiliani[4] made some observations upon the cadaver for the purpose of elaborating a practical method of reaching the chiasm, and, after trying various means of approach, he concluded that the frontal route was the only rational one. His operation, as practised on the cadaver, consisted in reflecting an osteoplastic flap of omega shape; the base of the flap was about one-half inch behind the coronary suture and three and a half inches broad, its width seven inches, and depth five inches. A similarly shaped flap of the dura was reflected after the longitudinal sinus, which at this point is no larger than a small vein, had been ligated. Elevation of the frontal lobe exposed the optic nerve.

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[4] Annals of Surgery, July, 1904.

This operation, though necessitating a very liberal exposure, need not be regarded as one of unusual gravity. The rapid and efficient methods of making an osteoplastic flap now in practice eliminate as a possible objection to this operation the magnitude of the flap. The success or failure of the operation would depend upon the ease with which *on the living subject, not on the cadaver,* the brain could be elevated sufficiently to expose the affected region. Though as yet I have had no experience, I would not hesitate in a suitable case at least to make the attempt to expose the tumor.

OPERATIONS UPON THE CEREBELLUM FOR RELIEF OF CHOKED DISKS.

One of the most constant symptoms of tumors of the cerebellum is choked disk. The appearance of certain subjective symptoms, such as headache and vertigo, may prompt the patient to consult a physician before he has noticed any disturbance of vision, but in most cases by the time the physician is consulted an examination of the eye-grounds will reveal choked disks of greater or less intensity.

Indications for Operation.—In general it may be said that indications for operation in cases of suspected tumor of the cerebellum do not differ materially from those that apply in tumors of the cerebrum. In both classes of cases, once the diagnosis has been made, operation should not be delayed for any length of time. Too much stress cannot be laid upon the importance of presenting the case to the surgeon at the earliest possible moment. Months have been spent in the fruitless administration of antisyphilitics before operation has been considered, and in many cases an unwarranted amount of time has been spent in the efforts to establish the diagnosis beyond the peradventure of doubt or to localize the tumor with mathematical accuracy. As Kocher has said, there is no more excuse to-day for delaying operation because the tumor has not been localized exactly than there would be for declining to operate upon a case of intracranial hemorrhage because one was unable to determine the exact seat of the clot. Postponement of operation should be discountenanced for several reasons. In the first place, in cases of long diminish the amount of hemorrhage. To afford greater freedom for the necessary steps of the operation, a suitable head-rest should be attached to the operating table (Fig. 4). Schede places his patients in the sitting posture leaning far forward. This posture, he says, diminishes hemorrhage to a considerable degree, but the position is an awkward and difficult one in which to retain the fully anæsthetized patient.

Incision.—The incision begins at the tip of the mastoid process on the affected side and runs parallel to and one centimetre above the superior curved line. From this point a vertical incision is made sufficiently long to expose the field of operation. The flap should contain the skin, muscle, and pericranium. If the pericranium and muscles are reflected together, there will be considerably less hemorrhage.

Removal of Bone.—For various reasons it is not feasible or desirable to make an osteoplastic flap. The muscles and aponeurosis are thick enough in this region to offer adequate protection to the underlying structures and to make bony closure of the opening unnecessary. A small opening is chiselled out at a point midway between the occipital protuberance and the mastoid process. Here the bone is comparatively thin and, as Poirier says, this is the point of greatest safety. When completed, the opening in the skull should extend upward one centimetre above the superior curved line, outward as far as one can go without opening the mastoid cells, inward to within one centimetre of the median line, and downward to a point at least one centimetre distant from the foramen magnum.

Exploration.—After the dural flap is reflected, one proceeds at once to search for a tumor, unless it has been decided to resort to the two-stage operation. The principles to be applied in deciding this question are precisely those which have been adopted in our operations for tumors of the cerebrum.[5] Protrusion of the cerebellum through the opening, once the dural flap is reflected, is indicative of increased intracranial tension and suggests the presence of a tumor, abscess, cyst, or internal hydrocephalus. Under normal conditions the cerebellum is under greater tension than the cerebrum, and, when the dura is incised, a normal cerebellum will protrude in many cases, at least to a slight degree. The surface of the cerebellum should be inspected and palpated on all sides, to discover, if possible, any abnormality in appearance or consistency. If there is reason to believe that the tumor is subcortical, an exploratory incision is made, and, if the neoplasm is found, it is removed. On the other hand, if the symptoms point to a tumor at the cerebellopontile

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[5] American Journal of the Medical Sciences, February, 1904.

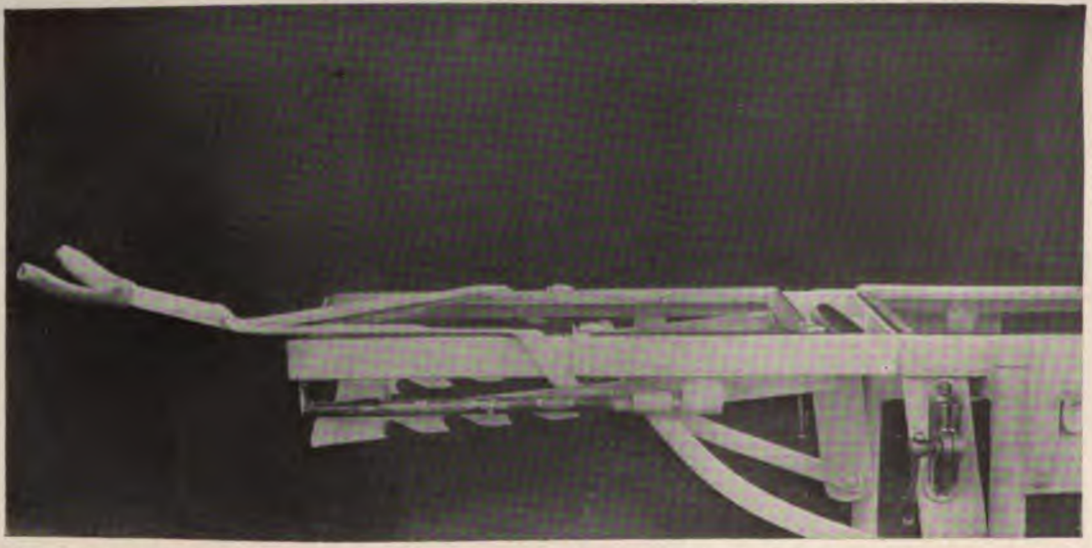

Fig. 4.—A headrest that may be used to advantage in operations upon the head. The device is a very simple one and does not require a screw or bolt to secure it in place.

angle, the subsequent steps of the operation will be attended with some difficulty.

Exposure of the Cerebellopontile Angle.—To enable one to expose a tumor in this region it may be necessary to resort to one of two procedures, tapping of the lateral ventricles or removal of a large portion of the cerebellar hemisphere. The former method is disapproved of, on

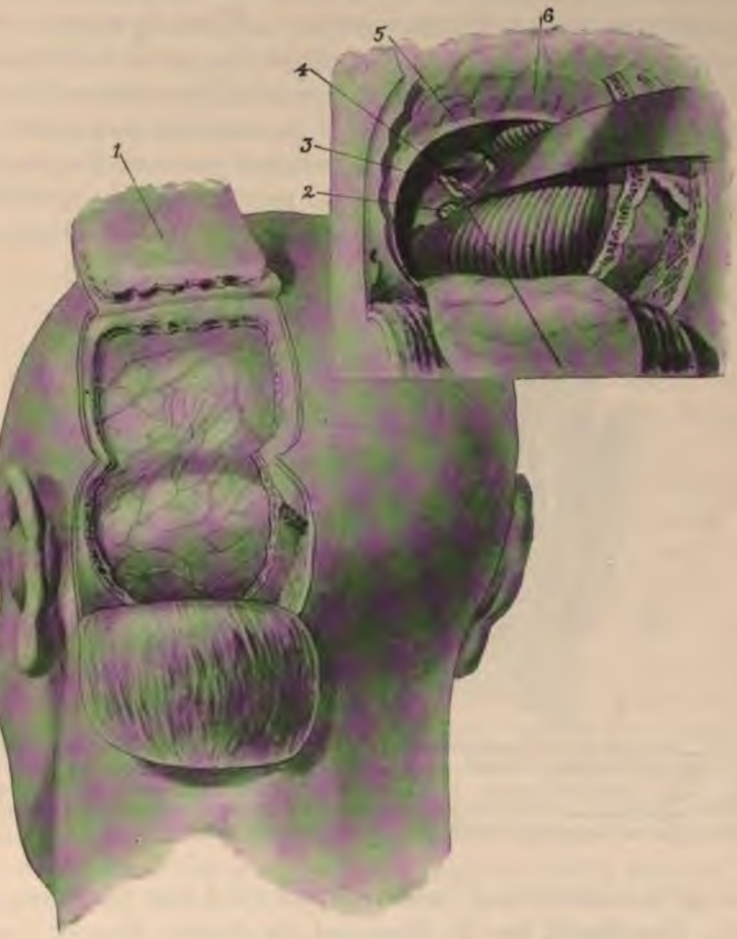

FIG. 5.—The larger figure to the left illustrates the operation for the combined exposure of one cerebellar hemisphere and the occipital lobe of the cerebrum. The smaller figure, above and to the right, illustrates the structures in relation to the anterior aspect of the cerebellum and the petrous portion of the temporal bone. Attention is called especially to the position of the fifth, seventh and eighth cranial nerves. This drawing was made by viewing the structures from the lateral aspect, such an exposure as would be made in exploring for tumors of the cerebellopontile angle. 1. Osteoplastic flap reflected in an operation for the combined exposure of occipital lobe and cerebellum. 2. Ninth, tenth, and eleventh cranial nerves. 3. Auditory nerve drawn to one side by retractor in order to expose. 4. The facial nerve which lies directly beneath it. 5. The root of the trigeminus as it enters the groove at the apex of the petrous portion of the temporal bone.

the ground that it is so fatal in its tendencies. The alternative, on the other hand, is attended with very different results. The impunity with which large collections of cerebellar tissue may be removed, not only without endangering life but without disturbance of function, was an observation made by physiologists long ago, and which has been confirmed in a number of cases in my own experience. When, therefore, it is difficult to expose the cerebellopontile angle, or if a tumor has been found and there is not sufficient space for its removal, the operator should not hesitate in removing at least a third of the cerebellar hemisphere (Fig. 5). The dangers attending the manipulations necessary for removal of a tumor from this region, owing to the proximity of the medulla and pons, are not imaginary, and it is important to avoid infliction of a degree of traumatism upon these structures which may determine a fatal result. One should bear in mind that the shortest distance from the surface of the skull to this angle, and, therefore, the best method of approach, is along a line parallel to the petrous portion of the temporal bone. The region to be exposed should be approached, therefore, from the lateral rather than the superior or inferior aspect of the cerebellum. Not only is this the shortest route, but the safest also, in that the manipulations are carried on at a point farthest distant from such vital structures as the pons and the medulla.

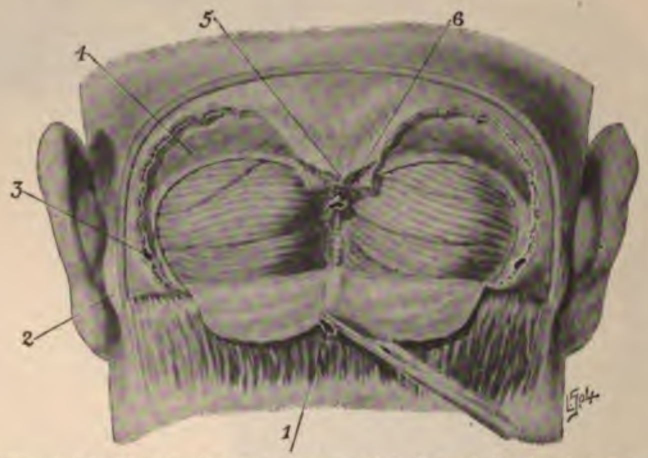

FIG. 6.—Operation for the simultaneous exposure of both cerebellar hemispheres, necessitating ligation of the occipital sinus. 1. The occipital sinus, which has been ligated previously and reflected with the dura. 2. Mastoid process. 3. A large tributary of the lateral sinus, invariably opened in cerebellar craniectomies and of varying dimensions; said to be sometimes as large as the lateral sinus. 4. Lateral sinus. 5. Occipital protuberance. 6. Occipital sinus.

Simultaneous Exposure of Both Hemispheres; Bilateral Craniectomy.—In many cases it will be impossible to determine beforehand whether the tumor is in the right or the left lobe, so that, failing to find the tumor on one side, one must proceed at once, or after a proper interval of time, to explore the opposite side. In deciding this question one must be governed entirely by the condition of the patient. The incision is extended from the termination of the point at the occipital protuberance to the opposite mastoid process and the flap reflected in the manner already described (Fig. 6). An opening in the bone is made in the same manner and of the same dimensions as on the opposite side and the intervening bridge of bone removed with a Gigli saw. The falx cerebelli is punctured on either side of the occipital sinus and the latter divided between two ligatures. This will enable one to reflect a flap of the dura covering both hemispheres and to displace the cerebellum with greater freedom than would be possible if an intervening bridge of bone remained between the two openings. Bilateral craniectomy should not be resorted to as a routine procedure, but should be restricted to those cases in which the tumor is believed to occupy a position near the mesial surface or in which the tumor was not found on the side first exposed. In most instances it should be practised in two sittings.

Palliative Operations.—Under certain circumstances we despair of being able to perform a radical operation: either the tumor cannot be found or cannot be localized; it may be inaccessible or it may have attained such proportions as to make its removal impracticable. In any of these contingencies a palliative operation is justifiable, and in some cases should be regarded as imperative. The headache, vertigo, and vomiting, so constant in cerebellar tumors, makes the life of the patient pitiable, and yet he may be relieved of all these symptoms for a considerable time by adopting such measures as will relieve pressure. But the strongest argument against delay of operation in the treatment of cerebellar tumors is the possibility of being able to save the patient's vision: choked disk is one of the most constant symptoms and, if permitted to continue too long unrelieved, an optic atrophy will develop, and the time for complete or even partial restoration of vision will have passed. Palliative operations upon the cerebellum do not consist alone in the removal of the overlying bone for the relief of tension, as in such operations upon the cerebrum, but in the removal of a portion—one-third to one-half—of the cerebellar hemisphere. Such a procedure, mutilating as it seems, does not appear to add to the gravity of the operation, nor does it cause any demonstrable disturbance of function, but affords that relief of pressure which alone will secure the desired results.

Hemorrhage.—Hemorrhage is not troublesome once the bone has been removed; occasionally, however, there may be an alarming hemorrhage from one or another of the various sinuses that traverse the occipital bone. In exceptional cases these sinuses are so large that a patient may lose an enormous amount of blood in a very short time, so that one should be prepared always with suitable means for controlling hemorrhage from this source. Horsley's wax should be tried first, but, if this fails, the outlet of the sinus should be closed with plugs of wood which can be whittled to the proper size and length from ordinary swab-sticks. Nothing but carelessness would permit of injury to the lateral sinus during operation. Should such an accident occur, it is quite feasible to control bleeding by ligation of the sinus.

Results.—My personal experience leads me to believe that the dangers attending cerebellar operations have been somewhat exaggerated. I have been struck especially with the comparatively slight depression attending these operations and with the rapidity with which reaction ensues. It should be borne in mind, however, that, when the tumor is found, the act of removing it is not of itself attended with any additional risks. Mention has already been made of the fact that patients with cerebellar tumors are not subjects favorable for operation, yet the risk lies not in any feature or stage of the operation, but in the operation *per se;* therefore, the mortality attending those cases in which the tumor has been found and removed should not be greater than that attending an operation which has been solely exploratory. The mortality will be reduced to a minimum by observing most scrupulously those measures which are said to be preventive of shock: securing prompt hæmostasis, inflicting a minimum degree of traumatism upon the cerebellar tissue, avoiding undue traction or pressure upon the pons and medulla, and resorting to the two-stage operation according to the principles already laid down.

The effects of the removal of tumors, or in palliative operations of the removal of a portion of the cerebellar hemisphere, are as prompt as they are gratifying. Headache and nausea cease almost at once; choked disk subsides, and vision may be almost restored or considerably improved within a few days; the ataxia disappears and the stability of gait is often restored. The results of operations upon the cerebellum, whether radical or palliative, are usually more striking and give a degree of relief greater than after similar operations upon the cerebrum. The following statistics were compiled from one hundred and sixteen cases of cerebellar tumors selected by the author:[6]

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[6] New York Medical Journal and Philadelphia Medical Journal, February 18, 1905.

CEREBELLAR TUMORS.

	Per cent.
Tumors found	45
Tumors not found	55
Removal with recovery	15
Removal with improvement	13.9
Removal without improvement	0.9
Improvement without removal	13.9
No improvement without removal	13.9
Death when tumor was removed	12.9
Death when tumor was not found and not removed	28.7

A comparison of the statistics of successive dates shows an increase in the percentage of recoveries and improvements and a reduction in the mortality:

Results.	Frazier's Table, 1904. Per cent	Duret's Table, 1903. Per cent.	Oppenheim's Table, 1902. Per cent.
Recovery	15	14	7.5
Improved	28	25	7.5
Unimproved	15	..	13
Mortality	42	60	71

A comparison of the statistics of the total number of cases in Frazier's collection with the statistics of the last five years shows a manifest improvement in the results:

Results.	Total Number of Cases. Per cent.	Cases reported during past five years,—1899-1904. Per cent.
Recovery	15	24
Improved	28	28.5
Unimproved	15	11
Mortality	42	25.8

SURGERY OF THE SEVENTH NERVE; OPERATIVE TREATMENT OF FACIAL PALSY.

Historical.—It has been proved by experimental work that regeneration in a partially degenerated nerve will occur when the peripheral segment of the affected nerve is united to the proximal segment of a perfectly healthy nerve. The first application of the principles here involved to the treatment of facial palsy is credited to Ballance and Stewart, who, in 1895, performed the first operation upon the human subject, uniting the facial to the spinal accessory nerve. Since that time this operation, modified in several ways, has been performed a number of times, with varying degrees of success.

Indications.—Not every case of facial palsy presents the indications for the operative treatment. To prevent the method from falling into disrepute, the cases should be selected with the utmost care. In the consideration of the treatment, cases of facial palsy for convenience sake may be classified as follows:

(1) Cases due to traumatism,—*e.g.*, gunshot and stab wounds or fracture of the base of the skull. (2) Cases due to an infective neuritis secondary to mastoiditis. (3) Cases due to Bell's palsy. (4) Cases of paralysis following section of the nerve in the removal of a tumor from the face (Ballance).

As to the traumatic cases, the sooner the operation is performed the better the prognosis; therefore, when the nerve is believed to be destroyed, the operation should be performed without delay.

In those cases secondary to mastoiditis operation should be resorted to as soon as the infection has subsided and the possibility of wound infection from that source eliminated.

Delay is not only justified but proper in all cases of Bell's palsy, but only for a limited time. If after the expiration of six months the paralysis has not responded to appropriate treatment (when the muscles are almost completely paralyzed and the reaction of degeneration is very pronounced), the operation should be performed at once. Does the operation hold out any hope to those who have suffered from this condition for years—two, five, fifteen, twenty years or more? In answer to this question it should be said that this is of comparatively little significance in so far as the nerve itself is concerned. The keynote to the situation is the condition of the muscles. If the facial muscles are completely atrophied and will respond no longer to galvanic stimulation, the prospects of restoration of function are extremely doubtful. Up to the present time seven years and nine months (Hackenbruch's case) is the longest period at the expiration of which the function of the nerve has been restored. Since the condition of the muscles plays such an important part in the prognosis, too much emphasis cannot be laid upon the importance of persisting in the employment of electricity and massage,—*i.e.*, such measures as will tend to preserve muscle tone.

As contraindications to the operation may be mentioned cases of congenital facial palsy, provided the palsy is the result of failure of development of the nerve and its muscles in the medulla oblongata, and cases of such long duration that the muscles are so wasted that there is little substance capable of contraction and the nerve is so atrophied as to render it improbable that it could be found.

Technic.—Before undertaking the operation two questions must be considered,—first, the selection of the nerve with which the anastomosis is to be effected, and, second, the method by which the anastomosis is to be made. The facial being a motor nerve, one should select as its anastomotic complement a motor or a mixed nerve. Two nerves have been proposed,—the spinal accessory and the hypoglossal. Of these the hypoglossal is to be preferred, chiefly because the disagreeable complication of associated shoulder movements is avoided and because the cortical centre of the tongue is situated nearer to the facial centre. After the facial nerve has been anastomosed to the spinal accessory there follows an association of the movements of the shoulder with those of the face,—that is to say, a voluntary or involuntary contraction of the muscles of the face is accompanied by a contraction of the muscles of the shoulder, and *vice versa.* In course of time these once conjoint movements become to a limited extent dissociated,—to the extent that the movements of the face may become independent. The patient may exercise control over certain groups of facial muscles independently of any associated movements of the shoulder, but, so far as I have been able to discover in the records of cases hitherto reported, there has not been a single instance in which a voluntary effort to move the shoulder was not attended with contraction of the facial muscles. These so-called associated movements constitute, without doubt, a very serious objection to the selection of the spinal accessory nerve.

Another argument in favor of the hypoglossal nerve has to do with the relative positions of the cortical centres (Fig. 7). The cortical centre of the tongue is situated nearer the facial centre than that presiding over movements of the shoulder. Whether it is true that dissociated movements of the face are matters of education of the newly established cortical centre or that the impulses that precipitated those movements originate in the old facial centre or in the tongue centre, the hypoglossal nerve should be chosen. If the first or "educational" theory is correct, the hypoglossal nerve should be preferred because the movements of the tongue are much more closely allied with those of the face than those of the shoulder, and it is reasonable to infer that the newly-selected cortical centre could for this reason be more easily and effectually educated to perform its new function; or if the second theory should be proved the correct one,—viz., that the impulses must originate in the original facial centre,—the tongue centre has the advantage over the shoulder centre in that the impulses would not have to travel as far from the tongue to the facial centre as from the shoulder to the tongue centre.

In this connection Spiller says, " In selecting the hypoglossal nerve we select one whose cortical centre is functionally more allied to that of the facial than is the centre of the spinal accessory, or, let us say, more accurately, than is the centre for the lateral movement of the head or elevation of the shoulder, inasmuch as movements and not muscles are represented in the cerebral cortex. Even if associated movements persist in the tongue after division of the hypoglossal,—and I cannot at present understand how they can do so if the motor supply of one side of the tongue is completely destroyed,—these movements would be hidden from observation, and would not cause the annoyance to the patient produced by spasm of the face every time the shoulder or upper limb on the same

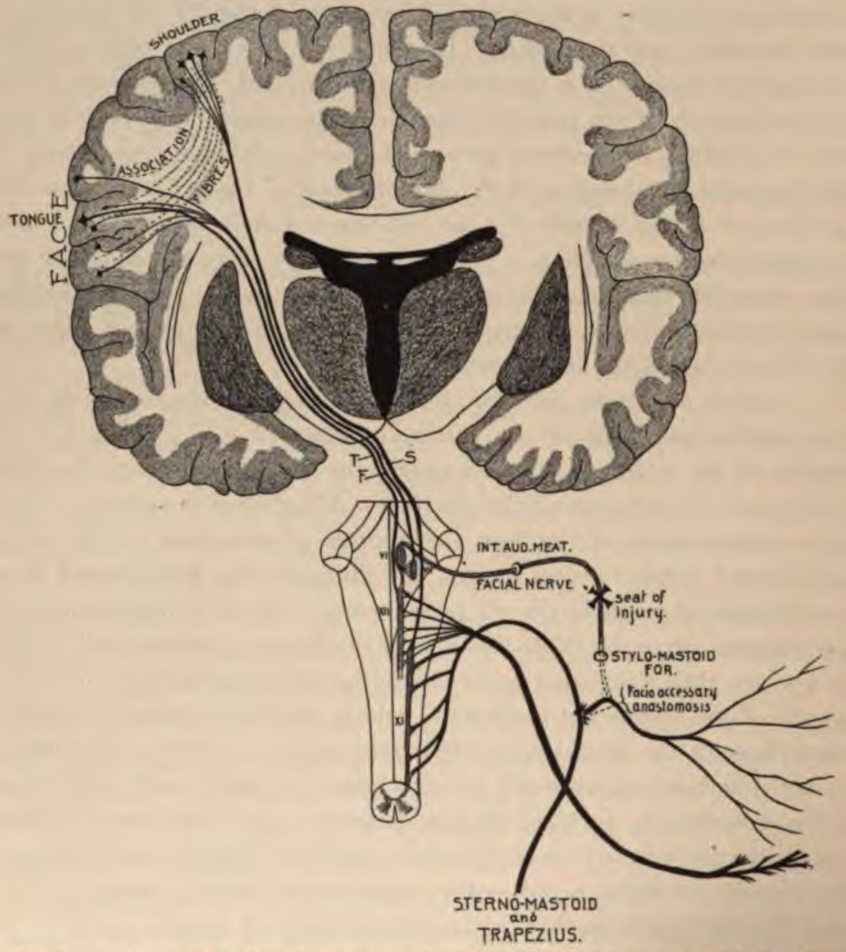

FIG. 7.—Showing cortical origin of nerves supplying muscles to face, tongue, and shoulder. (From Ballance and Stewart's article on the Operative Treatment of Facial Palsy, British Medical Journal, May 2, 1903.)

side is raised, after the spinal accessory has been employed for the anastomosis. Turning of the head or raising the shoulder is only partly controlled by the spinal accessory, and we can readily understand why an overflow into the anastomosed facial nerve occurs if the mov
which the spinal accessory normally has an important part i

The cortical centre for the spinal accessory being only a part of the centre for turning the head or raising the shoulder, I doubt very much whether this centre can become so educated that it can assume perfectly the function normally exercised by the centre for the movements of the face."

Of less significance is the question as to whether the nerve should be completely severed and the movements of the muscles it supplied sacrificed or whether only a portion of the nerve should be used to complete the anastomosis. If the spinal accessory was severed, there would be complete paralysis of the sternocleidomastoid and partial paralysis of the trapezius and drooping of the shoulder. Division of the hypoglossal will be followed by paralysis of the depressors and some of the elevators of the hyoid bone and with atrophy of one-half the tongue. As compared with the deformity which it is hoped will be relieved, either of these disturbances may be regarded as equally insignificant. An end-to-end anastomosis should be preferred to a lateral anastomosis, for the following reasons: in the first place, the operation is easier of execution, if one relies on the entire nerve-trunk and not a portion or a branch with which to make the anastomosis; second, because, if it is true that the selected cortical centre must be educated to preside over the facial muscles, the unlearning of its original and the learning of its new function will be facilitated if the connection is severed completely from the peripheral muscle which it supplied originally and from its accustomed centripetal excitation; lastly, as Spiller has pointed out, the chances of full restoration of power are greater after end-to-end anastomosis because the whole restorative force of the central end of the completely divided nerve is directed into the peripheral end of the facial.[7] No matter which view of regeneration is accepted, reunion of the divided nerves is absolutely essential to complete regeneration. If, therefore, only a part of the healthy nerve is brought in contact with the facial, we cannot expect so satisfactory results as when the entire transverse surface of the healthy nerve is sutured to the end of the facial stump.

No matter which nerve is selected, the operation is not difficult (Fig. 8). The facial nerve is exposed by hugging closely the mastoid process and displacing the parotid gland forward and outward (Fig. 9). The nerve will be seen entering the substance of the gland about one centimetre above and one centimetre to the inner side of the tip of the mastoid process. If the hypoglossal nerve has been chosen, it may be found crossing the external carotid artery just below the point at which the occipital artery is given off. The nerve is traced, freed, and finally divided at such a distance to the inner side of the external carotid artery as will enable the proximal end to be brought in contact with the peripheral stump of the facial without undue tension. The point of anastomosis will be upon the posterior belly of the digastric muscle. The spinal accessory nerve may be found at the junction of the sternocleidomastoid muscle and traced upward. In some instances the branch to the trapezius has been used instead of the main trunk.

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[7] If from clinical observation it is proved that restoration of function will occur equally well after a lateral anastomosis, the latter operation may be substituted for an end-to-end anastomosis, providing the stump of the facial nerve is long enough to reach the hypoglossal nerve. Taylor recommends that the nerve be divided as far up in the canal as possible, in order to render available a longer section of the nerve, and thus make it possible to bring the facial nerve in apposition to the hyp[illegible]e without undue tension.

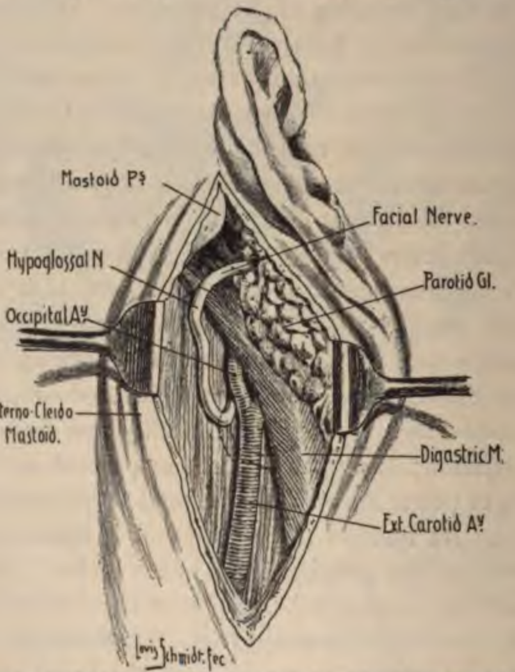
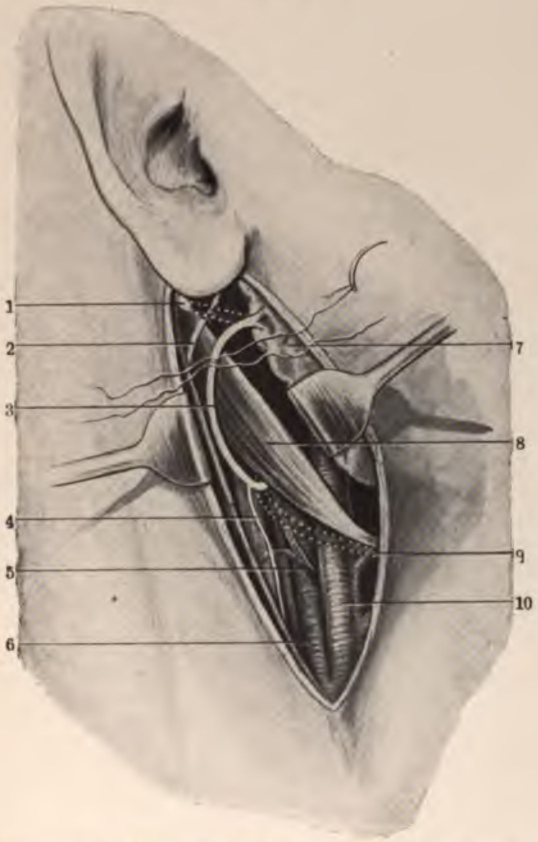


FIG. 9.—Author's operation for the facio-hypoglossal anastomosis.

In making an end-to-end anastomosis three or four sutures will suffice to effect apposition. These should be introduced only through the sheaths of the respective nerves, using for this purpose the finest curved needles and silk. In lateral or end-to-side anastomosis, either the end of the divided facial may be buried within the fibres of the hypoglossal or spinal accessory and secured in place by sutures, or, as recommended by Ballance and Stewart, a longitudinal incision may be made in the sheath

PLATE C.



Author's operation for anastomosis of the facial with the hypoglossal nerve.

1. Stump of the central segment of the facial nerve; 2. Spinal accessory nerve; 3. Reflected portion of the hypoglossal nerve; 4. Descendens noni; 5. Occipital artery; 6. Internal carotid; 7. Parotid gland drawn forwards with a retractor; 8. Digastric muscle; 9. Course of hypoglossal nerve before it was reflected; 10. External carotid artery.

of both nerves sufficient to expose the fibres, and the sutures so introduced through the sheaths of each nerve as to bring the two surfaces of exposed nerve-fibres into apposition.

Clark's method effects an anastomosis by embedding the pointed end of the facial nerve into the substance of the hypoglossal and securing it there with sutures (Fig. 10).

It is needless to say that with each of these methods success depends upon perfect union of the nerve, and this can only be obtained by proper coaptation and by providing every condition which will favor uncomplicated repair of the wound. Delicacy of manipulation, use of the minimum number of sutures, perfect apposition, and complete asepsis may be considered absolutely essential to ultimate success.

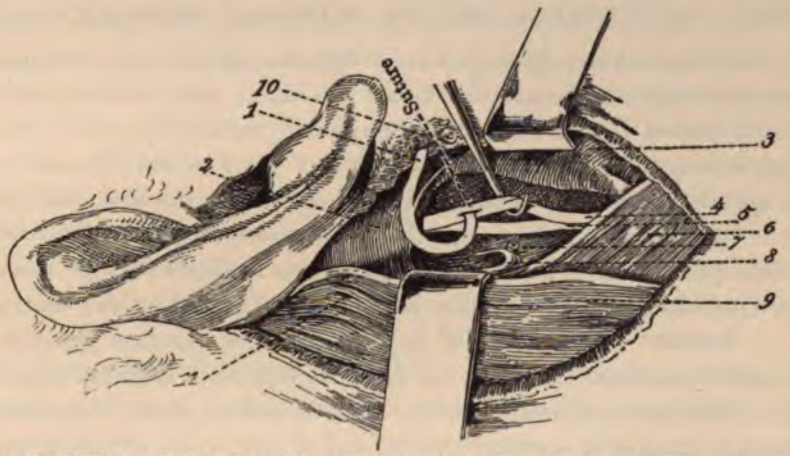


FIG. 10.—Clark's method of lateral anastomosis: 1, facial nerve; 2, styloid process; 3, internal jugular vein; 4, hypoglossal nerve; 5, pneumogastric nerve; 6, posterior belly of digastric muscle; 7, transverse process of atlas; 8, occipital artery; 9, sterno-mastoid muscle; 10, parotid gland; 11, mastoid process.

Results.—There may be said to be three grades or degrees of improvement, leading to complete recovery, which may be anticipated after the performance of this operation. The first and the least degree of improvement which could be expected is the restoration of muscular tone: the muscles are no longer flaccid; the face in repose is symmetrical,—not, as before, asymmetrical. If no greater improvement was anticipated, the prospect of obliterating the deformity in repose should settle any doubt in the mind of the patient as to the advisability of undergoing or in the mind of the surgeon as to the advisability of recommending the operation. The second stage or grade of improvement, and one which has been obtained in a majority of cases, is the voluntary control of individual muscles or groups of muscles, such as those concerned in raising the eyebrows, in closing the lids, in puckering the lips, or in whistling. Such restoration is a very material gain, for not only is the patient's face symmetrical in repose but in action as well. The third or last grade, the attainment of which signifies complete recovery, is the return of the expressional or emotional movements, as laughing, crying, etc. The mechanism of these movements is much more complicated and a matter of delicate co-ordination. This is the ideal result, but one which has as yet not been obtained. Symmetry of face in repose, symmetry of the face in certain uncomplicated movements, and, lastly, symmetry in the more elaborate movements, as those of expression—these are the improvements aimed at in the surgical treatment of facial palsy. (For further remarks on facial palsy see Chapter VII, p. 332.)

VISUAL DISTURBANCE DUE TO INTERNAL HYDROCEPHALUS.

The acquired or secondary form of hydrocephalus is very frequently associated with disturbance of vision; in fact, optic neuritis and its results, choked disk and atrophy, are almost invariably present. Bitemporal hemianopsia may be present, too, as a result of the pressure upon the middle portion of the optic chiasm exerted by the distended third ventricle. These conditions may be relieved only by the withdrawal of cerebrospinal fluid. To this end one of two methods may be resorted to,—Quincke's lumbar puncture and puncture of the lateral ventricles

1. **Lumbar Puncture.**—Of the two operations lumbar puncture is the safer procedure, although the fatal tendencies of both are considerable. Gumprecht collected fifteen cases of sudden death after lumbar puncture in cases of cerebral tumors, and of eight cases of lumbar puncture employed in cerebellar tumors four died within a few days. The sudden disturbance of pressure is no doubt responsible for the large majority of fatalities. Furbinger attributes death to pressure exerted upon the bulb by the arrest of cerebrospinal fluid from the ventricles at the foramen of Magendie, and it is no doubt especially dangerous when the communication between the lateral ventricles and subarachnoid space and the canal of the spinal cord is interrupted. Under these conditions the equilibrium between the cerebral and spinal fluid is disturbed; the medulla and pons are drawn forcibly downward into the foramen magnum, with resulting traumatism, followed by serious if not fatal consequences. When the communication with the lateral ventricles is cut off, lumbar puncture will, of course, be ineffective. The results of this treatment for the relief of chronic hydrocephalus are on the whole unsatisfactory. The condition may be ameliorated temporarily by the relief of pressure, but only in the rarest instances will the results be

permanent. Gowers, for example, secured a permanent result in a case of serous meningitis of the chronic type; the headache, vomiting, ataxia, and paralysis of the ocular muscles completely disappeared, and the optic neuritis subsided. Oppenheim recommends lumbar puncture for those cases in which the tumor is associated with an internal hydrocephalus and especially when the tumor encroaches upon the posterior fossa and threatens the life of the patient. To withdraw fluid from the lower portion of the dural sac a hollow needle from two and a half to three inches long, armed with a stilet, is introduced between the second and third lumbar vertebræ. With the patient in the recumbent posture and the back strongly arched, the needle is introduced at a point about five millimetres to the right of the median line, on a level with the space between the second and third spinous processes. The needle is directed slightly inward and upward until the dural canal is reached. It is not necessary to aspirate the fluid, as there is enough pressure in the canal to force the fluid out. In children the needle may be introduced exactly in the median line just below the spinous process of the second lumbar vertebra. Whenever it is desirable to determine the pressure under which the cerebrospinal fluid exists, a U-shaped manometer may be attached by a rubber tube to the needle. The manometer may be used to advantage in all cases as a guide to the amount of fluid which can with safety be withdrawn. A pressure of twenty millimetres of water should be preserved.

2. **Ventricular Puncture.**—Puncture of one of the lateral ventricles has been performed for the relief of hydrocephalus, but, as in the case of lumbar puncture, the effect will be only palliative. This is an operation which is, unfortunately, of unusual gravity, and the danger attending it is so great, in comparison to the possible benefit, as to make it a procedure of questionable propriety. Many cases have been reported in which the results were disastrous. So far as I have been able to find, there has been but one instance in which the treatment was successful. The lateral ventricle may be entered from the frontal region, as recommended by von Bergmann, or from the parietal region, as recommended by Keen. By the former method a small longitudinal incision is made a little in front and to the inner side of the tuberosity of the frontal bone. A hole is bored through the skull and a long, hollow needle is introduced in a direction from before backward and a little downward and inward and pushed steadily into the brain tissue until the fluid escapes. In Keen's method the needle is introduced thirty-two millimetres above a line connecting the supraorbital edge with the external occipital protuberance and thirty-two millimetres behind the external auditory meatus.

SURGERY OF THE FIFTH NERVE; TRIFACIAL NEURALGIA; TIC DOULOUREUX.

In the vast majority of cases tic douloureux is a disease of the second and third divisions. In many cases the pain appears first in the distribution of the third division and may remain so for a long time before spreading to the branches of the second division. Of these two divisions the inferior dental and the infraorbital nerves are the branches which are almost invariably involved. In exceptional cases the neuralgia may begin in the first or ophthalmic division and never extend to the other branches; on the other hand, the pain may be referred to the branches of all three divisions. In addition to the neuralgic pains we find in some cases certain points (" tender points") along the course of the nerve which are sensitive to pressure. Thus, when the first division is involved, the " tender points" will be found at the supraorbital notch, in the outer part of the upper eyelid, and at the emergence of the nasal branch at the lower edge of the nasal bone; the pain radiates over the anterior half of the head, to the eyelid and eye itself, and to half of the nose; the second division gives off a few branches as it traverses the sphenomaxillary fossa and appears at the infraorbital foramen as the infraorbital nerve, where it divides into the palpebral, nasal, and labial branches. When this division is involved, the " tender points" will be found at the infraorbital foramen, at the side of the nose, and over the most prominent portions of the malar bone. If the dental branches are involved, pain will be referred also to the teeth and gums of the upper jaw. Finally, if the third division is involved, pain will be referred to the distribution of one or all of its sensory branches; to the parietal eminence, temple, and external ear (temporal and auricular temporal branch), to the tongue (lingual), to the gums and teeth of the lower jaw (inferior dental branch), and to the lower lip (mental branch). The " tender points" are at the inferior dental foramen, where the auriculo-temporal crosses the zygoma, over the parietal eminence, in the tongue, and at the mental foramen.

The character and intensity of the pain are very variable. As in neuralgias of other nerves, the pain is spontaneous and paroxysmal; a series of successive pains constitute a paroxysm and several paroxysms an attack. The pain is variously described as darting, stabbing, sharp, pricking, tearing, and boring; as to intensity, the pains vary from a slight burning sensation to one of excruciating severity. The attacks are sometimes initiated by premonitory signs, such as a feeling of tension, itching, or a feeling of unrest in the facial muscles, and they are precipitated in some instances by a draught or exposure, by talking, chewing, swallowing, by fatigue and emotion. There is the widest variation in the frequency with which attacks recur: there may be an interval of only a few hours, or several months; but usually as time goes on the attacks increase both in frequency and severity. There can be said to be no periodicity except in those cases in which the neuralgia is of malignant origin or in which the attacks occur only during the catamenial periods. (For further remarks on fifth nerve see Chap. VII, p. 320.)

Concomitant Symptoms.—Associated with the neuralgic attacks there may be signs of irritation of the secretory, trophic, and vasomotor branches as well as of the motor root and the facial nerve. Thus, one may see lachrymation, flushing, or sweating (vasomotor disturbances); loss of hair and hair-pigment, and herpes (trophic disturbances); contraction of masseter muscles (motor root), and convulsive movements of the face (facial nerve). The pulse is sometimes accelerated, sometimes excited and irritable.

Pathology.—In order to comprehend the various clinical manifestations of trifacial neuralgia and the rationale upon which the indications for treatment are based, one should be familiar with at least certain elementary facts relating to the pathology of the affection. There may be said to be two or three separate and distinct types of tic douloureux; one in which the lesion is primarily a neuritis of the peripheral branches, which in course of time may or may not extend to and involve the ganglion; another in which the ganglion is affected primarily; and a third, perhaps, in which the lesion is neither in the ganglion nor in its branches, but in the central nervous system. In some instances the process is a degeneration, in others an interstitial neuritis, or the primary lesion may be a neoplasm. Were it possible to determine by the clinical manifestations whether the lesion were central or peripheral, the indications for treatment would be more clearly defined. Sometimes, however, it is possible. According to Wagner it may be assumed that a neuralgia is of cerebral origin providing only several branches are involved, and providing, too, that in the other cranial nerves there is further evidence of cerebral disease.

Prognosis.—Trifacial neuralgia is of itself not a fatal disease, although if unrelieved the patient may from the pain and suffering become exhausted and more easily fall a prey to some intercurrent affection. In some instances the disease may be said to be self-limited and without any operative intervention may gradually disappear. This is more especially true of the so-called migrainous type, which is observed in women in early life suffering from migraine. The prognosis

[illegible]

Classification — [illegible]

[illegible]

the disease has a tendency to run its course in five or six years and then exhibits a tendency to spontaneous cure. The drug which is *par excellence* the most efficacious, especially in the anæmic and exhausted, is strychnia. In cases of but one or two years' standing strychnia properly administered will arrest or control the disease almost invariably. To obtain the best results the drug must be administered in heroic doses, and the patient should be kept under the closest observation, and should be confined to bed. The remedy is administered hypodermically once daily, in ascending doses, until, at the expiration of two weeks, the physiological limit is reached. Beginning with one-thirtieth grain the dose may be increased to one-fourth to one-half grain, and when the maximum dose is reached it should not be given oftener than on alternate days. Quinine, arsenic, potassium iodide, and aconitine nitrate have been recommended, but not one of these drugs compares in efficacy with strychnia.

Extracranial Operations.—These operations include the so-called peripheral operations and those which attack the nerves at their exit from their respective foramina at the base of the skull. As to the indications for this class of operations the general statement may be made that in every case in which the pain seems to be confined to a branch of one of the three divisions of the ganglion a peripheral operation is called for. The simplicity of such a procedure, as compared with an operation at the base of the skull, whether extra- or intracranial, should deter us from subjecting the patient to the additional risk of more serious procedures, even taking into consideration the fact that there may be a recurrence. The tendency of the peripheral nerves to regenerate after a neurotomy or even a partial neurectomy should be combated by an extensive extirpation of the nerves, thus reducing to a minimum the chances of recurrence.

Given a case in which the pain may be referred to more than one branch of but one division, it is proper under such circumstances to resort to an operation upon the nerve trunk immediately after its exit from the foramen. For example, in neuralgia of the second division, a neurectomy just above Meckel's ganglion would be called for. For those cases, however, in which at least two of the three divisions are believed to be involved one or the other of the intracranial operations should be resorted to.

Peripheral Operations.—At one time it was thought sufficient simply to expose and divide the nerve. Owing to the natural tendency of the nerve to regenerate, recurrences were frequent. At the present time a peripheral operation should not be considered properly executed unless either by excision or avulsion a considerable section of nerve is removed. The longer the section removed the less the likelihood of regeneration, and, therefore, of recurrence.

With this idea Thiersch devised a method of forcible extraction, which consisted in grasping the nerve with forceps and making gradual traction upon the nerve both centrally and peripherally by turning the forceps on their own axis. For this purpose the blades of the forceps are so constructed that they do not cut through the nerve-fibres. One blade of the forceps is concave, one convex, and both are ribbed transversely or longitudinally, care being taken not to have any sharp edges. After the nerve is dissected free from its surroundings it is grasped at right angles to its axis. Traction is made very slowly, not making more than one-half turn per second. By this method not only may a greater portion of the peripheral segment even to its small terminal filaments be extracted, but the central segment of the nerve up to the ganglion.

According to van Gehuchten avulsion or the forcible extraction of the peripheral nerves will be followed by degeneration not only in the ganglionic cells but also in the bulbospinal root. If this result was constant, restoration of function and recurrences of the pain would be impossible. Thus far, with the exception of a few cases, the results are based upon animal experimentation, but they are sufficiently positive to warrant the adoption of Thiersch and van Gehuchten's suggestion in all peripheral operations.

Exposure of the Supraorbital Nerve.—To reach this nerve an incision is made parallel with the superior margin of the orbit for an equal distance on either side of the supraorbital foramen. The periosteum is divided and the nerve will be exposed to view and should be traced back to a point behind that at which the supratrochlear branch is given off.

Exposure of the Infraorbital Nerve.—The infraorbital foramen from which the nerve makes its exit is situated about one centimetre below the inferior margin of the orbit and about the junction of its middle and inner third. A line drawn from the supraorbital notch downward between the two lower bicuspid teeth intersects the infraorbital and mental foramina (Keen). The foramen may be exposed by a short incision down through the skin, subcutaneous fat and the attachment of the levator labii superioris. The periosteum is divided and separated below sufficiently to expose the plexus and above sufficiently to expose the floor of the orbit. The roof of the canal is so thin that it is easily broken open with a groove director. The nerve is now grasped with forceps and by means of traction or with the aid of the scissors resected back as far as the sphenomaxillary fissure.

Exposure of the Inferior Dental Nerve.—The inferior dental nerve enters the canal at a point about three centimetres above the angle of the jaw. In order to avoid injuring the branches of the facial nerve, or Steno's duct, and to give the best cosmetic results, the incision should be made just beneath the lower border of the jaw. A musculo-periosteal flap, including the masseter, is reflected upwards and the canal opened with a chisel or trephine. In order to guard against regeneration the nerve should be resected as far forward as the mental foramen or forcibly extracted by the Thiersch method. As a further safeguard against recurrence the author plugs the central opening of the canal with some impermeable substance, as silver foil.

Extracranial Operation at the Base of the Skull.—These operations are reserved for those cases in which recurrence of pain has followed one or more peripheral operations upon the branches of the second or third division, as infraorbital or inferior dental nerves, and especially when pain is referred to the entire area of one or the other of these two divisions. If both divisions are involved, an intracranial operation should be performed.

Second Division.—To reach the second division at the base of the skull one must enter the sphenomaxillary fossa by traversing the antrum (Carnochan's method) or by a resection of the malar bone (Lücke's method). Neither of these operations is difficult of execution, although the latter is to be preferred; it affords more readily a better exposure and avoids the possibility of wound-contamination from the antrum of Highmore.

Method of Lücke.—Beginning one centimetre above the outer canthus and two-thirds millimetre from the external orbital margin an oblique incision is made, terminating at a point corresponding to the position of the upper third molar. At right angles to the first and starting at its upper end a second incision is made so as to expose the zygomatic process of the temporal bone. The malar bone is divided in the line of the first incision with a chain or wire saw and the zygomatic arch is cut through at and in front of the articular tubercle. The osteoplastic flap thus formed, consisting of skin, zygomatic arch, and masseter, is now reflected. Following the posterior surface of the upper jaw the fat and plexus of veins are displaced backwards; in this way both the venous plexus and the internal maxillary artery may be protected from injury. Pressing on now, in the direction of the sphenomaxillary fossa, and removing as much as may be necessary to afford adequate exposure, the operator will reach the superior maxillary nerve at a depth of about six centimetres in its passage from the foramen rotundum across the sphenomaxillary fossa to the infraorbital canal. Before proceeding to extract the nerve it must be dissected free from the infraorbital artery. It is then grasped with a pair of forceps, divided close to the foramen, and its peripheral portion extracted by the Thiersch method. Friedländer resects the entire malar bone, and, while a better view is afforded, the operation is more radical, and there is always the possibility of opening the antrum of Highmore.

Chavasse's Modification of Carnochan's Method.—A horizontal incision is made below the eye and from the centre of this a vertical incision is dropped, exposing the infraorbital nerve. An opening an inch in diameter is made with trephine or chisel in the anterior wall of the antrum and in the posterior wall a somewhat smaller one is similarly made. The nerve is grasped with a pair of forceps and divided at the infraorbital foramen; by breaking through the floor of the orbit the nerve may now be traced through the antrum and sphenomaxillary fossa back to the foramen rotundum, where it is divided with curved scissors. Precautions must be taken not to injure the internal maxillary artery which lies in close relation with the posterior wall of the antrum.

Third Division.—This division, with all its branches, the lingual, inferior dental, buccinator, and auriculo-temporal nerves, may be exposed by Krönlein's method. Beginning one centimetre from the angle of the mouth an incision is made to a point one centimetre in front of the lobule of the ear. The buccinator muscle is left undisturbed, but the incision is carried through that portion of the masseter not covered by the parotid. Steno's duct and the branches of the facial nerve should not be injured. After separating the attachments of the masseter and internal pterygoid muscles from the coronoid process the latter is divided obliquely forward and downward and as low down as possible. The osteoplastic flap composed of the resected coronoid process and the temporal branches is reflected and the inferior dental nerve is isolated and traced back to the foramen rotundum, where the main trunk is divided. To enable one to isolate the nerve it may be necessary to dissect away the fatty tissue of the cheek, anterior to the masseter muscle, as well as the more deep-seated fat in relation with the lateral aspect of the internal pterygoid muscle.

Intracranial Operations.—*Division of the Sensory Root of the Gasserian Ganglion; Physiological Extirpation of the Ganglion.*—Upon the assumption that roots of the central nervous system, when divided, do not undergo regeneration, Spiller, in 1899, proposed division of the sensory root as a substitute for excision of the ganglion for the treatment of trifacial neuralgia. Before putting the idea into practice upon the human subject a series of experiments were conducted by Spiller and the author upon dogs, from which it was concluded that it has yet to be proven that regeneration of the central roots can occur. With the assurance derived from this experimental evidence the first operation was conducted upon the human subject in 1901. In this case the symptoms were relieved, and there has been no recurrence. Since then the operation has been practised a number of times with equally gratifying results.

This operation was proposed by Spiller with the hope that it would be found to be easier of execution than excision of the ganglion, and from our clinical experience it may be said that this expectation has been fully realized. The advantages of this procedure over any other having for its object the removal of the ganglion may be summed up as follows: (1) It is easier of execution. The most difficult step of the operation for excision of the ganglion is not undertaken until the attempt is made to free the ganglion from its base. To divide the sensory root the ganglion is left undisturbed once the dura propria is reflected from its superior aspect. All that remains to complete the operation is the division of the root; thus before the most difficult stage of one operation is even approached the other operation is finished. (2) There is less hemorrhage. The most annoying feature of these operations as a class is hemorrhage, and the most troublesome hemorrhage begins when one starts to elevate the base of the ganglion, since the ganglion receives its largest blood supply from vessels at the base. In division of the sensory root hemorrhage from this source does not occur because the base of the ganglion is not disturbed. (3) The adjacent structures are not exposed to injury. The proximity of the cavernous sinus, and of the third, fourth and sixth nerves to the inner aspect of the ganglion exposes them to injury whenever the attempt is made to extract the ganglion; in point of fact the cavernous sinus has been frequently torn and one of the nerves divided in extraction of the ganglion. (4) Ocular disturbances, such as corneal ulceration, are less likely to occur. This is probably due to the fact that the sympathetic fibres, which pass through the ganglion, exercise a trophic influence on the eye. In division of the sensory root these sympathetic fibres are not disturbed. (5) The operation is easier of execution. (6) It should be attended with a lower mortality. It may be assumed that of two operations the safer would be the one in which there is less hemorrhage, fewer technical difficulties, and, therefore, greater economy of time.

Technic.—The technic of the operation is precisely the same as that for the extraction of the ganglion up to the point at which the superior and posterior aspects of the ganglion have been exposed. At this juncture the sensory root is picked up on a small blunt hook, grasped with a hæmostatic forceps, and a small section excised.[8]

Excision of the Gasserian Ganglion.—Although it has been said that the ganglion may be reached readily through the occipital fossa, the best method of approach unquestionably is through the temporal region, as advocated first by Hartley and a few months later by Krause.[9]

Although the operator must be thoroughly familiar with the anatomy of the structures concerned, it would not be amiss to call attention here to some points of considerable practical importance. In the first place, an attempt should be made to avoid injuring the branch of the facial nerve which supplies the orbicularis palpebrarum. The inability to close the eye becomes a very serious matter after operations in which there is always the possibility of corneal ulceration. The incision usually recommended for this operation is so placed that this branch of the facial nerve will be cut almost invariably. Cushing suggested, therefore, that the flap be so outlined that the integrity of the nerve be not endangered. Second, one should be familiar with the course of the middle meningeal artery, and also with the fact that in some cases, in its passage across the anterior inferior angle of the parietal bone the artery is encased in a bony canal. The superior margin of the flap should be low enough to avoid injuring the vessel at this point. Otherwise the artery would inevitably be torn. This is not a serious complication; but would delay the operation at the most only a few moments. The foramen spinosum is easily identified by tracing the middle meningeal artery to the base of the skull. The position of this foramen is important on account of its relation to the ganglion and its branches. The foramen lies a little external to the posterior aspect of the ganglion and a little behind and external to the foramen ovale through which passes the third division. It is, therefore, an important landmark, and a knowledge of its whereabouts will be especially helpful to those who have had limited experience in this field of surgery. Finally, mention should be made of the intimate relation of the third, fourth, and sixth cranial nerves, as well as of the cavernous sinus, with the internal aspects of the ganglion.

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[8] The early and subsequent stages of the operation are described in the technic for excision of the ganglion.

[9] No one should undertake the operation for the first time upon the living subject. The operation should be repeated several times upon the cadaver in order that at least some of the difficulties may be appreciated and a vivid impression of the exact anatomical relations acquired.

The Operation.—A horseshoe-shaped incision is made with its base corresponding to the level of the zygoma and its summit three or four centimetres above. The musculo-cutaneous flap is reflected and the zygoma resected. The latter step is important, as it enables the operator to approach the ganglion from a plane on a level with the base of the skull; the nearer one is to the base of the skull the shorter the distance to be traversed before the ganglion is reached. With mallet and gouge an opening is made in the skull and then further enlarged with rongeur forceps until the opening measures three or four centimetres in diameter. With a blunt instrument, as the handle of a knife, one then proceeds to separate the dura from the base of the skull, following in a general way the direction of the petrous portion of the temporal bone. At this stage of the operation hemorrhage may be a troublesome though never an alarming feature. The firmer the adherence of the dura to the skull, and, as a rule, the older the patient the freer will be the hemorrhage. Thus it may be necessary from time to time to stop to control hemorrhage by packing firmly with strips of gauze and allowing the same to remain *in situ* for one to three minutes.

When the foramen spinosum is reached the operator separates the dura anterior to the foramen until the second and third divisions are exposed as they pass into the foramina rotunda and ovale, respectively. The field of operation during this and the subsequent stages of the operation can only be exposed to view by elevating the tempero-sphenoidal lobe, and for this purpose a suitable retractor should be provided. The exposure of the ganglion is never difficult, although hemorrhage may be so free as to necessitate one's proceeding rather slowly. The most difficult step of the operation is now approached. To thoroughly isolate the ganglion it must be separated from its dura propria; to accomplish this an incision is made with an Allis director or other blunt instrument into the dura from the second to the third division, and by blunt dissection the superior aspect of the ganglion is exposed. After dividing the second and third divisions the ganglion is separated from its base, grasped with a pair of forceps and forcibly extracted. In doing this the cavernous sinus and one of the contiguous cranial nerves have been torn. Hemorrhage from the lacerated sinus may be controlled by packing, and if the middle meningeal artery has been injured near the foramen spinosum bleeding may be checked by ligating the vessel by plugging the foramen or by gauze packing. The wound is irrigated with normal saline solution and a strip of rubber tissue introduced for drainage. Certain precautionary measures should be adopted to protect the eye and prevent corneal ulceration. Immediately before or after the operation a Buller bandage should be applied and the eye irrigated twice daily with a warm boracic solution. In addition to this Keen sews the eyelids together before the operation is begun, and does not remove the stitches until the third day. The immediate results of the operation will vary considerably according to the experience of the operator. There is no field of surgery for which there is a greater justification in specialization than in the surgery of the central nervous system. The mortality of Gasserian ganglion operations has been estimated as 12 per cent.; but there is no doubt that with greater attention to those requirements of technic which have been elaborated only in recent years, and especially in the hands of those who have had exceptional opportunities, the mortality will be reduced.

Abbe's Method.—As a substitute for excision of the ganglion Abbe recommended the forcible extraction of the second and third divisions and the interposition of a piece of gutta-percha tissue one and one-half inches by three-quarters of an inch to prevent degeneration. His technic differs somewhat from that already described in that he practises preliminary ligation of the external carotid artery and exposes the bone by a vertical incision over the middle of the zygoma. Two objections to this method of treatment at once suggest themselves: the first, that the operation is applicable only to those cases in which the pain is distributed only in the second and third divisions; the second that there is a possibility of the rubber tissue acting in the rôle of a foreign body setting up a reaction in the tissues, which would terminate in abscess formation and necessitate the removal of the foreign material. Abbe says: " It is certainly past dispute that there is no need for the removal of the first branch of the fifth pair in any case of grave tic douloureux unless the origin is to be found in a tumor of the Gasserian ganglion or behind it." This may apply to the majority of cases, but it certainly is not a rule without exception. In fact, in two cases under my own observation the pain was most intense in the distribution of the first division. It might also be said of Abbe's operation that it offers no assurance against subsequent involvement of the first division, and, so far as the mortality is concerned, it should be no less than that following division of the sensory root, since both are intracranial operations, the only difference being that in one two peripheral branches are divided, while in the other one divides the central root of the ganglion.

Osmic Acid Injection.—While Billroth and Neuber were the first to employ osmic acid in the treatment of trifacial neuralgia, they used repeated injections in the skin without exposing the nerve. Burnett (1899) was the first to recommend the more careful intraneural injections into the exposed nerve, and more recently (1904) Murphy has become an advocate of this mode of treatment. Up to the present time, however, it has not been proven that the results are much better than would be expected after an excision of the nerve trunk. The injection of osmic acid no doubt makes it more difficult for the nerve to undergo regeneration, but as yet we have no assurance that regeneration will not eventually occur. Besides, cases have been reported [10] in which the treatment was a failure. The immediate results, as regards the harmlessness, and at least the temporary relief, might be said to justify the use of this treatment as a substitute neurectomy, before resorting to procedures of such severity as intracranial operations. It can in no wise, however, be regarded as a substitute for the intracranial operations in those cases of major tic in which the pain is widely distributed. As to the technic, a one and one-half to two per cent. solution of osmic acid should be injected in many places into the exposed nerve trunk and also into the foramen. Murphy has injected the palatine, mandibular, lingual, supraorbital and infraorbital branches. The injections should be made under general anæsthesia, and all of the branches, except the supraorbital, can be exposed through mouth incisions.

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[10] Wright, Medical Chronicle, Fourth Series, vol. vi.

CHAPTER XXII.

TREMORS; REFLEXES; GAITS.

BY JOSEPH SAILER, M.D.

TREMORS.

BY tremor we mean an oscillatory movement of some part of the body, occurring as a result of more or less rhythmical muscular contractions, which alternate in opposed groups of muscles.

Carefully to be excluded are all the forms of pseudotremor produced by rhythmical shocks occurring within the body, such as Musset's sign in aortic regurgitation, certain movements occurring as a result of aneurism, and movements in the extremities which are the result of vascular pulsation. There is some doubt as to whether fibrillary contractions within a certain muscle or group of muscles which do not produce movements at any of the joints, or waves of contraction passing along the muscles, should be included under tremors or not.

Tremors are not necessarily signs of morbid conditions. They may occur as a result of certain physiological states. Thus, tremors of the extremities of more or less severity occur during and after prolonged severe exertion, in the course of violent emotional disturbance, after chilling, the most frequent example being chattering of the teeth, after excesses of various kinds, and during convalescence from severe infectious disease. They may also be produced voluntarily, either for purposes of simulation or experimentation. In all these forms the movement is slow and rhythmical, and the duration is short. The head and limbs are the parts most commonly involved, and the movements are at least partially under the control of the will.

Among the causes of pathological tremor may be mentioned particularly the *poisons*. Alcohol, especially chronic alcoholism, is apt to be associated with a fine tremor of the hands, lips and tongue. Tobacco is said to cause a fine tremor of the hands and eyelids. Lead, mercury, anilin, and various other poisonous substances, may also produce tremors. These are nearly all fine, slow tremors, often increased by voluntary movement, and affecting chiefly the upper extremities and the muscles of the face. In the course of various diseases of the nervous system characteristic tremors occur that aid materially in the diagnosis.

CLASSIFICATION.

Tremors are classed, first, as rhythmical and arhythmical. The rhythmical tremors frequently show in addition a rhythmical increase and decrease in their severity. We also speak of fine and coarse tremors. In the former the oscillation is very slight, perhaps imperceptible; in the latter it is of greater amplitude. Tremors may be regarded as rapid when the rate of oscillation is from eight to twelve times per second, and slow when it is less than five times per second. Intermediate forms also occur. Tremors are spoken of as static if they are present during rest, and as movement or intention tremors if they are either produced or increased by voluntary movements. If the tremor resembles some familiar motion it is usually called by that name, as the tremor of negation when the head is rotated rhythmically from side to side; the nodding or salaam tremor when the head oscillates forward and backward, and the pill-roller's tremor of paralysis agitans.

Numerous efforts have been made to obtain some information regarding the nature of tremors by means of recording apparatus. These all consist essentially of a moving surface and some transmitting mechanism by means of which the oscillations are recorded. It has been possible by these methods to determine the rhythm, the rate, and, in a relative manner, the amplitude of the oscillations, but practically nothing more, and the studies have not contributed materially to our knowledge of disease of the nervous system.

It will be most convenient to discuss briefly those tremors which occur in association with nervous disease, and are therefore of more or less diagnostic significance. There is one condition in which the tremor is practically the only sign of a morbid process. This is the simple hereditary or congenital tremor. It occurs sometimes sporadically, sometimes in several members of the same family, and occasionally exists in successive generations. The tremor is usually rather slow in rate and fairly rhythmical. It may affect almost any part of the body, but as a rule the head or upper extremities are particularly involved. The tremor sometimes develops only during activity or emotional stress, but often continues during rest, and indeed may be most pronounced during rest. Occasionally, but not often, it is under voluntary control. It may possibly be regarded as a stigma of degeneration, if this term has any real significance, but it is often found in intellectual persons, and even when it involves the upper extremities, does not appear to interfere with occupations requiring delicate manipulations. Usually it does not develop until adolescence, or adult life, but occasionally it is present in infants (Raymond).

constant tremor of the extremities and head, not much affected by movement, nearly always disappearing during sleep, slightly arhythmic and moderately rapid. The tremor is frequently unilateral, and is to be distinguished as hysterical only by the presence of the other stigmata of that condition. In common with all hysterical stigmata, it may be temporarily or permanently abolished by some profound emotional shock. Hysterical patients may also manifest from time to time irregular waving motions, sometimes tremor-like, but often so slow that they cannot properly be included under this designation. They may resemble gestures, and occasionally have a distinctly histrionic character. They often have, in addition, a rapid, violent tremor that may be produced by different attitudes, movements, or even associations. This tremor may affect one extremity, the side, or perhaps the whole body. The movements are rhythmic, rapid, and of considerable amplitude. They usually increase in violence until the particular action that causes them to cease is applied. This may be either the assumption of some attitude on the part of the patient, pressure upon the trembling extremity, or some other purely suggestive influence. Occasionally, however, the tremor increases until it becomes actually a clonic convulsion, or it may terminate in a hysterical crisis. Tremors of this type resemble very closely the clonic spasms of various conditions, particularly epilepsy.

The third group are the imitation tremors. Krafft-Ebing called particular attention to the frequency with which hysterical patients simulate the tremor of paralysis agitans, and various other types of tremor in organic disease are also imitated more or less closely. It seems to be common to all these tremors that they are more in evidence when the patient is under observation than when apparently alone; that their amplitude is greater than the real organic tremors, that they are not likely to be rhythmic; and that they do not cease as a result of the conditions that cause the cessation of the organic tremors.

In **neurasthenia** a fine tremor of the extremities, static in character, may often be detected. In **epilepsy** a tremor of the hands has been noted during periods of disturbance of consciousness that replace the convulsive attack.

In **general paresis** there is a fine tremor of the tongue and lips and occasionally a slight tremor of the hands. In hemiplegia and in other forms of spastic paralysis tremors are very apt to occur. They are usually coarse, irregular, and sometimes closely simulate the movements of ataxia. They are nearly always of the movement or intention type, and are exaggerated by emotional disturbances. Often with a return of power the tremor gradually improves and may ultimately entirely cease. Occasionally a very fine tremor persists in the side that has been paralyzed, even after an apparent complete return of power.

Akin to tremors are the choreiform movements, the movements that occur in ataxia, and various types of clonic spasm, as, for example, ankle-clonus. Their description, however, will be found in other sections of this book.

REFLEXES.

By the term "reflex" we understand some manifestation of vital activity that occurs as a result of obvious stimulation. Possibly all forms of vital activity are the result of stimulants of one kind or another, and therefore, properly speaking, reflexes, but unless the stimulus can be detected we are not justified in using this term. Clinically, we make a distinction between the psychical and the mechanical reflexes. By the former we mean the various actions that occur as a result of the stimulation of the perceptive centres of the brain, of which an example is the secretion of the saliva at the sight of food. Such reflexes have to do rather with psychology than with clinical medicine, and will not be further considered in this article.

By a mechanical reflex we understand the involuntary contraction of a muscle or group of muscles as a result of the stimulation of certain sensory fibres. This definition implies that the stimulus is transmitted along the sensory fibre to some part of the central nervous system in which there is a path of communication between the receptive sensory cells and the emissive motor cells, and then from the motor cells along the motor nerve-fibres to the muscle or muscles involved. This is spoken of as a reflex arc, and it is considered that whenever its continuity is broken the reflex necessarily ceases to occur.

Theoretically a sharp distinction is drawn between reflexes and muscular contractions which result from direct stimulation of the muscle involved, or of its motor-nerve supply, but in clinical work this distinction cannot always be maintained, and there are not wanting investigators who contend that many of the reflexes upon which most dependence is placed in diagnosis, including, for example, the knee-jerk, are the result rather of direct irritation of the muscle, either by sudden elongation or by some other method, than of the completion of a reflex arc by the primary stimulation of the peripheries of the sensory nerves. It must, of course, be understood that the integrity of the nerve supply of the muscle by which its tone is maintained is essential to the production of the normal contraction. The distinction between the two theories is more philosophical than practical. Muscular contractions can, of course, also be produced by the direct irritation of motor nerves, either at their termination or in the course of the nerve-trunk, as, for example, the contraction of the little and ring-fingers which occurs when the ulnar nerve is forcibly pinched. It is customary to speak of a reflex whenever, as a result of mechanical stimulation, particularly in the form of percussion, or stroking, a motor response occurs. The number of reflexes is already considerable, and every year three or four additions are made, most of them unimportant. They are classified in various ways: sometimes anatomically, as reflexes of the head, trunk, or extremities; sometimes according to the part to which the sensory stimulation is applied, as the skin, periosteum, bone, and tendon reflexes; sometimes into the skin or "superficial" and the "deep" reflexes.

The mechanism of the reflex action has been, ever since the first description of the knee reflex by Erb and Westphal, a matter of painstaking investigation, and a considerable number of theories have been evolved. Taking the knee-jerk as the type of the deep reflexes the following facts appear to have been definitely ascertained:

The reflex is best elicited when the muscle is moderately stretched. It varies in general as the muscle tone; but this rule is subject to many exceptions, in states of hypotonia exaggerated reflexes being present (Muskens). It is modified by various external conditions, thus, attention to the reflex diminishes it; distraction of the patient's attention by either psychical or motor occupation increases it. It is usually increased as a result of fatigue or cachexia. Any diminution of the functional activity of the sensory fibres leading to the lumbar portion of the spinal cord diminishes or abolishes the reflex. Complete destruction of the motor fibres leading from the lumbar portion of the cord to the quadriceps extensor also abolishes the reflex. Partial destruction of the peripheral motor neurones may be compatible with the persistence of the reflex, and even some degree of exaggeration, as in amyotrophic lateral sclerosis. Destruction of the centre for the reflex in the lumbar cord causes abolition of the reflex. Complete destruction of a segment or segments of the spinal cord above this centre usually causes loss of the reflex, but occasionally it persists, and is even exaggerated. If the posterior columns of the spinal cord are destroyed, as, for example, in tabes dorsalis, it is abolished. If the lateral pyramidal columns are destroyed it is greatly increased. Lesions in the brain involving the motor centres commonly cause a great exaggeration. Lesions not involving the motor centres usually cause a moderate exaggeration, but the reflex may be lost if the lesion is situated in the frontal lobes or in the cerebellum.

It appears from an analysis of these facts that the mechanism of the reflex involves more than a sensory and a motor fibre and the connecting link in the spinal cord. Therefore it has been customary to assume the existence of a second arc, the afferent fibres being the continuation upward of the sensory fibres passing from the periphery to the cord, and the efferent fibres being the axis cylinders of the upper motor neurones, the arc being completed by some of the commissural fibres of the brain. This second arc appears to have a double function; that is to say, the persistence of the sensory part is necessary to the persistence of the reflex; the loss of the motor portion tends to enhance the reflex. It must be remembered, however, that the destruction of the sensory portion some distance above the entrance of the nerve fibres into the cord occasionally may occur without loss of the reflexes, and if this injury to the sensory portion occurs above the nuclei of the columns of Goll and Burdach in the medulla, the loss rarely occurs.

It is of course difficult to understand how the two limbs of an arc could exhibit so much difference in action. It may be assumed, for the sake of argument, that the upper arc is not really an arc at all; that the motor fibres passing from the cortex are concerned—with reference only to reflex action—merely with the control of muscle tone, and particularly with the inhibition of involuntary movements (*Dreifaserschema*). Therefore, if for any reason, this inhibitory influence is removed, the reflex, of necessity, becomes exaggerated. It is more difficult to understand why the destruction of the sensory columns should diminish or abolish the reflex. It is supposed, for example, that this destruction always involves the peripheral sensory nerves that lead to the lower reflex arc. But this does not accord with the observation so frequently made that it can now be regarded as thoroughly established, that complete or even partial transverse lesion of the spinal cord in the upper dorsal or lower cervical portion is usually but not invariably associated with loss of the reflexes, which is not due merely to loss of sensation. Numerous explanations have been suggested, according to which this loss is not due so much to the destruction of the sensory columns above the point of lesion as it is to a degeneration of the peripheral motor neurones and of the cells of Clarke's column, which has been found in the segments in which the reflex arc is completed, or to an œdema of the lower part of the spinal cord inhibiting its functional activity, or to loss of certain influences emanating from the cerebrum or the cerebellum, or to disturbance of the circulation in the lower part of the cord leading to degeneration of the ganglion cells, or to the effect of shock upon the lower part of the cord, although the reflexes in the part above the lesion are not affected; or to degeneration of the posterior lumbar roots, this is called tertiary degeneration (Brasch); or to hypotonia. Van Gehuchten suggests that there may be increased inhibitory action. Aside from the degeneration of the cells in Clarke's column, no histological evidence has been discovered of injury to the sensory neurones. There appears to be some relation between the amount of injury done to the cord and the duration of the inhibition of the reflexes, at least in dogs. This loss is associated with a highly developed nervous system, for, according to Sherrington, in the dog and cat transverse sections of the cord cause exaggeration of the knee-jerks, and sometimes in human beings and in the ape the loss is not permanent.

The way in which the sensory stimulation occurs has been the subject of some dispute. In all the cutaneous reflexes it appears to be due to the stimulation of the cutaneous sensory nerves, whether that stimulation is perceived in consciousness or not. In the periosteal reflexes it is supposed that the sensory nerves in the periosteum receive and transmit the impulse. In the case of the simple muscular contractions due to direct irritation of the substance of the muscle, or its fascia, reflex action probably does not occur, or plays a subordinate rôle, and the same is true of muscular contractions due to irritation of a nerve trunk such as occurs in Chvostek's phenomenon. The dispute, however, has referred particularly to the tendon reflexes. The reflex occurs because a sharp blow is struck upon the moderately stretched superficial tendon of the muscle. Tendons are so poorly supplied with nerves, and particularly sensory nerves, that it does not seem possible that the reflex is a result of direct irritation. Therefore, it has been surmised that it is due to the irritation of the overlying skin, the underlying periosteum (both of which hypotheses may be dismissed, particularly the former, as the reflexes occur in conditions of anæsthesia of the thigh or if the tendon is exposed); or to stimulation of the sensory nerves in the muscle by vibrations communicated to it, or by its simple sudden elongation. There can be no doubt, with reference to the knee-jerk, at least, that a distinct, powerful sensory impression occurs, sufficient—when the inhibitory influence of the motor fibres is removed—to cause contraction not only of the muscles affected by the blow upon the patellar tendon, unimuscular, but also of other groups of muscles in the same leg, multimuscular or even in the opposite leg. When this occurs it is spoken of as a crossed reflex. From the fact that degeneration of the posterior columns of the cord inhibits the reflex, it seems not unlikely that the fibres of muscular sense are the ones chiefly concerned in the production of the so-called tendon reflexes. But the impossibility of separating these fibres in the nerve trunks renders all views upon this subject entirely hypothetical.

The essential nature of reflex action is not clearly understood. Reflexes were originally supposed to be involuntary movements of escape. Thus, the drawing up of the leg when the sole of the foot is tickled is apparently an attempt to remove the part from the source of the irritation. But it is impossible to explain all reflexes in this manner. It has been supposed that reflex action was the muscular response to a local irritation, psychology indicating that in the vast majority of cases an external stimulus precedes muscular activity, and that the psychical stimulus which may occasionally replace this represents merely stored-up external sensory impressions. According to this view, all action is essentially reflex in nature, but some of the reflexes are comparatively simple and quite regular, whilst others are complex, irregular, and apparently purposeful. Although this view may be entirely satisfactory to the psychologist, it is of no particular advantage to the clinician. He is compelled to regard certain activities produced by sensory stimuli as reflexes upon whose existence or alteration diagnoses may be based. All other forms of movement, if of clinical significance, must be considered from a totally different standpoint. It is therefore from a clinician that we have received the most ingenious and, in some respects, most satisfactory explanation. Von Strümpell believes that these reflex movements are vestiges of the complex coordinated reflexes of the lower animals, which in human beings, as a result of the increase in the cerebral motor activities at the expense of the spinal motor activities, have been reduced to insignificant and apparently purposeless movements. Therefore, he believes that when the cerebral controlling influence is removed, the reflexes become exaggerated. As a matter of fact, the reflex interval is shorter in apes and idiots than in normal persons (Silbertini). Of course it is necessary to omit from consideration all those movements due either to direct irritation of the muscles or to direct irritation of the motor nerves, which are not properly reflexes, but merely manifestations of the inherent and essential contractility of muscular substance.

Unfortunately, there is still considerable dispute regarding the division in which many of the reflexes should be classified; some holding that they are due merely to direct irritation of the muscle or its tendon; others that they are true reflexes following the sensory nerve to the central nervous system, and returning along the course of a motor nerve. It is not impossible that in a few instances the reflexes may be mixed, or at least in health may be merely muscular contractions, but when the inhibitory influence of the central motor mechanism is removed become true reflex movements.

In studying reflexes for the purpose of utilizing the knowledge obtained for diagnosis or prognosis, certain factors must be considered: (1) The condition of the patient at the time the reflex is tested; this involves not merely the physical, but also the mental condition, states of excitement, fright, or preoccupation often having considerable influence upon reflex activity. (2) The temperature of the room. (3) The mechanism employed for eliciting the reflex. (4) The position of the patient. With regard to the reflex itself, it should be noted (1) whether it occurs promptly or slowly; (2) whether the response is normal, exaggerated, or diminished; (3) whether other muscles than those usually concerned are involved in the response, and (4) the effect of the investigation upon the patient.

In general it may be said that conditions causing increased myotatic irritability increase reflex activity. Mental emotion, such as fright, often inhibits reflex action. Great excitement, on the other hand, may enhance it. Chilliness increases it, and extreme cold, sufficient to numb the peripheral nerves, appears either to diminish or abolish it. In sleep it is diminished or even absent (Rosenbach).

Nearly all reflexes may be elicited by the hand, but a great variety of instruments have been devised for the purpose of demonstrating them more readily. The most generally useful is the rubber percussion hammer which has a triangular piece of firm white rubber for its head, the blow being struck with the apex of the triangle. The ordinary percussion hammer may also be employed. Instruments have also been devised particularly for measuring the knee-jerk, so that the blows struck upon the tendon shall be uniform in force. It may be said in reference to all these instruments that up to the present time no clinical method for measuring reflex action has been devised that has such a degree of accuracy as to be in the least useful, and it is still necessary to estimate all reflexes according to the scale of "diminished, normal, or exaggerated," the determination to which one of these three classes any given reflex belongs depending entirely upon the judgment of the physician. Doubtless many errors are made, but on the whole the results have been of great service. It is possible that the method of Pandy for measuring the force of the knee-jerk may be of clinical value for the detection of marked paresis.

For the purpose of description, the reflexes will here be considered according to a convenient rather than a logical classification. The reflexes of the head, upper and lower extremities, and of the trunk will be grouped into tendon, cutaneous, and muscle reflexes. No attempt will be made to treat exhaustively those forms which are of doubtful nature and little or no clinical significance.

REFLEXES OF THE HEAD.

There are no tendon reflexes in the head. The other forms of reflex action, aside from the pupillary reflexes, that may be elicited are:

The Supraorbital Reflex.—This reflex was described by McCarthy in 1900 and consists of a slight, brief twitching of the orbicularis palpebrarum, most pronounced in the outer half, when a blow is struck upon the trunk of the supraorbital nerve. The reflex disappears in cases of destructive injury to the supraorbital nerve, causing anæsthesia in its area of distribution, and in peripheral facial paralysis. In cases of injury to the central nervous system no characteristic alteration usually occurs. A crossed supraorbital reflex may develop (Sailer). This may occur either under normal conditions, in which the twitching of the eye on the opposite side is very slight, or in cases of peripheral facial paralysis, in which it is quite distinct. Occasionally other movements occur, such as twitching of the ala of the nose or lifting of the corner of the mouth. These phenomena, however, are much more common in connection with the malar reflex. It has been claimed that the supraorbital reflex is not a true reflex, but is due to percussion upon the fibres of the orbicularis palpebrarum that spread upward from the eyebrow (von Bechterew). The crossed reflex is explained by assuming that the fibres on the opposite side may interlace. It seems difficult, however, to account for the disappearance of the reflex in cases in which the supraorbital nerve has been cut or injured (McCarthy). The chief clinical significance of this reflex appears to be that when it is present it indicates that the supraorbital nerve on that side is intact, and that there is no peripheral or facial palsy. When it is absent it indicates that either the supraorbital or facial nerve has ceased for some reason to convey impulses. As facial paralysis is usually easily detected, when it is absent the lesion can be localized to the supraorbital nerve. Of course, when present it may be uncertain whether the supraorbital nerve is affected or not. Under these circumstances, the existence of the crossed reflex would serve to indicate that the supraorbital nerve upon the paralyzed side is intact.

Chvostek's phenomenon consists of a sudden twitching of the muscles of the corner of the mouth, ala of the nose, and the external portion of the palpebral fissure when the trunk of the facial nerve is struck just after it winds around the posterior ramus of the lower jaw. This is not a true reflex, but simply exalted susceptibility of the nerve trunk to external stimuli. In certain cases this may be so great that percussion merely upon one of the branches of the nerve will suffice to produce the muscular reaction. Occasionally simple percussion upon the malar bone or pressure upon the eyeball (Fuchs) produces the same effect. This reflex is strictly pathological, and, as far as we know, pathognomonic of tetany, in all forms of which it may occur. Its absence does not exclude the existence of tetany, particularly in those forms grouped together as non-idiopathic; that is, associated with various other conditions, such as cachexia thyreopriva, pregnancy, dilatation of the stomach, etc. It is, however, regarded as of such importance that when it is absent it is usual to speak of the "incomplete type."

The malar reflex is either a muscle or a periosteal reflex, my opinion inclining strongly to the former view. In normal conditions percussion over the malar bone may cause a slight twitching of the upper angle of the mouth on the same side, but ordinarily produces no muscular response. In cases of recent facial paralysis of peripheral origin the myotatic irritability of the degenerating muscles is exalted, and percussion at this point causes contraction of the elevator of the upper angle of the mouth and movements of the ala of the nose. If the degeneration—as is usually the case—is at all pronounced, the movements will be exceedingly sluggish, the angle of the mouth moving up slowly, and slowly resuming its previous position. In facial palsy of central origin the malar reflex occurs more frequently than in health, and the movements are much quicker. Clinically, the presence of the malar reflex in peripheral facial paralysis merely indicates that the muscle is not wholly degenerated, a fact which can, of course, be ascertained by the electric current.

The chin-jerk is produced by tapping downward upon the teeth when the lower jaw is relaxed and partially open. For this purpose either a small flat object, such as tongue-depressor, may be rested upon the lower teeth, or the finger placed over the mental prominence. If the reflex occurs there is a sharp upward movement of the lower jaw. It appears doubtful whether this is a reflex. It is unquestionably exaggerated in cases of myotatic irritation, such as cachexia, and I have obtained it in deep ether anæsthesia. It is also likely to be increased in functional nervous conditions, such as hysteria with exaggeration of the reflexes. It does not appear to be altered in any characteristic way as a result of cerebral lesions, and bilateral peripheral lesions of the motor nerves that would be involved (facial, trigeminal) are so rare that there are no recorded observations regarding their influence upon it. It may, therefore, be regarded as of no clinical significance. It may also be elicited by tapping upon the insertion of the masseter (von Bechterew).

Other reflexes of the head involve particularly irritations arising from the mucous membrane in the nose and throat. These include the various nasal and pharyngeal and palatal reflexes. Their discussion, however, does not come within the scope of this article. Von Sölder has described a lateral movement of the jaw when the cornea is irritated. Pressure upon the eyeball through the eyelid may produce the same effect (Fuchs).

REFLEXES OF THE UPPER EXTREMITY.

The bicipital reflex is a tendon reflex that is obtained by striking upon the radial tendon of the biceps, producing a contraction of the muscle and slight lifting of the upper arm. The reflex is best elicited in the following manner. The forearm is flexed upon the arm at little more than a right angle. The arm is then grasped just above the elbow in such a manner that the thumb rests upon the biceps tendon. This is then slightly compressed and a blow struck upon the dorsum of the thumb. It is often convenient to allow the arm of the subject to rest upon that of the examiner. The biceps is a true tendon reflex. Its arc appears to consist of the sensory nerve-fibres passing from the muscle to the fourth and fifth cervical segments of the spinal cord, and then downward along the brachial plexus and the musculo-cutaneous nerve. The reflex is not invariably present in health. It is usually exaggerated in functional nervous conditions associated with a general increase in the reflex activity, and is almost invariably increased in lesions of the brain involving the motor mechanism of the arm. It is lost in lesions of the posterior columns of the cervical cord, as in late tabes dorsalis or in the cervical form of this disease; possibly even more constantly than the patellar reflex. It is also lost in cases of destruction of the peripheral nerves, such as injury to the brachial plexus. It diminishes in proportion to the wasting of the muscle in the various forms of myopathy that involve the muscles of the arms. Owing to its occasional absence in perfectly healthy individuals, inability to obtain it is not of great clinical significance unless it is unilateral. Exaggeration of this reflex, if associated with paresis or paralysis of the arm, may be regarded as a definite sign of injury to the motor portion of the cerebral hemisphere of the opposite side. Bicipital and tricipital clonus are very rare.

The tricipital reflex consists of contraction of the triceps muscle when its tendon is struck a sharp blow. It is best elicited by bending the forearm upon the arm at about a right angle and then striking just above the prominence of the olecranon. If the reflex is present there will be a sharp contraction of the muscle and extension of the arm. Care must be taken not to strike upon the muscle. This reflex is in all respects closely analogous to the biceps and is subject to the same modifications by various lesions. It occurs in about two-thirds of all healthy persons.

The supinator reflex is a contraction of the supinator muscle when a blow is struck upon the tendon as it passes over the radial bone. It is best elicited by having the forearm flexed upon the arm and at rest. There is usually slight flexion and supination of the arm. There is some doubt as to whether this is really a tendon and not a periosteal reflex. It is absent in about one-eighth of all healthy persons, is likely to be exaggerated in functional nervous disease, and is occasionally exaggerated on the paralyzed side in cerebral hemiplegia. It is of little clinical significance.

Striking upon the extensor or flexor muscles of the forearm causes them to contract, giving rise, respectively, to extension and flexion of the fingers. This is not a reflex, but merely an expression of muscular irritability, the reaction occurring most distinctly in states of cachexia, and in conditions in which the muscles are easily stimulated mechanically.

The hypothenar reflex consists of a contraction of the abductor of the little finger when pressure is made over the pisiform bone or when the end of the little finger is pinched. The contraction is usually tonic in character and is indicated by the appearance of a groove on the ulnar side of the hand. Occasionally there are a series of light twitches in the tonically contracted muscle. It is not constant, and nothing definite is known regarding the conditions that cause it to be increased—if such a change could possibly be recognized—or to be lost. I have observed it in conditions of anæsthesia of the hand, such as unilateral anæsthesia from hysteria, and in anæsthesia associated with hemiplegia of cerebral origin.

In some cases a *palmar reflex* may be obtained.

REFLEXES OF THE LOWER EXTREMITY.

Tendon Reflexes.—**The knee-jerk** is a contraction of the quadriceps tendon in response to a blow struck upon the patellar tendon, and was first described by Erb and Westphal. Clinically it is the most important of all the reflexes, and, as one of the first known, has been most extensively studied. It may be elicited in a variety of ways. I patient is lying on the back the hand should be placed under the and lifted until the angle formed by the thigh and leg is about A sharp blow is then struck upon the patellar tendon either witl finger, the side of the hand, or a percussion hammer. There is a su contraction of the quadriceps which causes the patella to move up and, if sufficiently vigorous, raises the foot slightly from the bed as sult of a forward kick. If the patient is sitting in a chair one thigh be thrown over the other and the leg allowed to hang loosely. A is then struck as before and almost always results in a slight for kick. If the patient sits on the edge of a table and allows the le swing the reflex can often be obtained very readily. In certain of extreme muscular atrophy, as a result of emaciation, the tendon be so loose that the reflex cannot be obtained in any of the ordi positions. Under these circumstances, if the leg is flexed str upon the thigh, it may be possible to stretch the tendon suffici to obtain a slight muscular contraction when it is struck. If the is so diminished that it cannot be obtained by any of these proce it may be *re-enforced* according to the method of Jendrassik. depends essentially upon the fact that anything in the form of cular or intellectual effort increases the vigor of the reflex. It is t fore customary to give the patient some simple task to perform, for example, as an attempt to pull apart the hands with the fi locked together. At the same time he should be instructed to look from the knee. Sometimes it is sufficient to give him a simple c lation in mental arithmetic. A convenient modification is to have grasp a dynamometer or the physician's left hand; the physician co "one, two, three," the grasp and the blow being performed as near possible at the same moment.

After the stimulus is applied there is perceptible interval befor muscular contraction occurs, which is called the latent period or interval. It has been measured by numerous observers working a great variety of instruments and in normal persons is probably 0.03 second. It varies considerably, however, in different individ and in the same individual under different circumstances. It is s ened by conditions that increase reflex activity; and by mental c tions that diminish inhibition, microcephaly, mania and epil The duration of the contraction is also variable (0.1–0.5 second) is greatly increased in spastic conditions. The curve of the mus contracture has also been studied, but has yielded no informatic importance regarding reflexes.

In testing the knee-jerk it is not sufficient merely to watch the excursion of the leg. Not infrequently, if the muscles are weak, no movement in the leg is produced, and yet the reflex may be actually exaggerated, as is shown not merely by the violent contraction of the muscle, but also by the involvement of other groups of muscles in the thigh. It is therefore always advisable to rest one hand upon the middle of the upper surface of the thigh. Occasionally the reflex can be detected by placing the finger upon the tendon and striking a blow upon the finger, the twitching produced by the muscle being then readily perceived. Instruments have been constructed for measuring the knee-jerk. They consist of an arc having approximately the radius of the length of the leg, with an indicator that is thrust forward by the kick of the foot. As it has been explained that the kick is not proportionate to the activity of the reflex, it will be obvious that none of them is of any value. At most they may serve to indicate an increased degree in the activity of the reflex in the same individual, and therefore may be useful in experimental work to determine the effect of various influences. It is always advisable, but rarely convenient, to test the knee-jerk with the thighs bare. Very frequently there is slight twitching of the adductors on the same side, and occasionally of the adductors or of the quadriceps on the opposite side. This is nearly always true if there is much exaggeration of the reflex. In estimating the reflex we have no standard of measurement, and are accustomed to use the terms "normal," "diminished," "absent," "increased," and "greatly increased" for the purpose of indicating roughly its degree. Unfortunately, the degree of variation is considerable in different individuals, and even at different times in the same individual. The following points may be of some value in the estimation.

The reflex is *absent* if, when the muscles are completely relaxed, and the patient is intelligent enough to employ the method of Jendrassik, and the physician is certain that the blow is struck over the patellar tendon,—that is, between the lower edge of the patella and the tubercle of the tibia,—no contraction of the muscles takes place. It is desirable that the leg should be carefully inspected, and at the same time that the free hand should be laid over the muscles of the thigh in order to perceive a contraction that might be invisible.

The reflex is certainly *diminished* if under ordinary conditions it cannot be obtained, and the response is slight when re-enforcement is employed. A visible response in a subject who has not previously been tested cannot be regarded as an indication of any pathological process.

The knee-jerk is certainly *exaggerated* if a slight tap upon the tendon causes the leg to fly out in an almost straight line; frequently, under these circumstances, two or more distinct twitches may be felt in the quadriceps muscle; if other muscles of the thigh, or of the opposite thigh, are involved; if there is patellar clonus; if the latent period is shortened and the duration of the muscular contraction lengthened, or if it is elicited by tapping on the periosteum of the tibia.

The knee-jerk is said to occur in all normal individuals. There is no doubt, however, that the limits of variation are very wide. In some persons apparently normal there are times when it can only be obtained by re-enforcement. In others it is invariably abnormally active. It is subject to great variation in disease. It is usually lost in cases of disease of the posterior columns, as, for example, in tabes and Friedreich's ataxia. In tabes it is absent on both sides in about 70 per cent. of all cases, and in about 15 per cent. more is pathologically altered. It may, however, even when lost, reappear in the later stages of the disease. It is lost in cases of total destruction of the peripheral nerves. It is reduced proportionately to the degeneration of the muscles in the various forms of myopathy. It is lost in cases of degeneration of the ganglion cells in the anterior cornua of the cord that control the quadriceps muscle. In partial injury of the peripheral nerves, such as occurs in multiple neuritis, it is nearly always absent or greatly diminished; but in a few instances, whose nature is not clearly understood, it may be exaggerated. It is somewhat variable, also, in total transverse lesion of the cord. In the majority of recorded cases it has been entirely abolished by total transverse lesions in the dorsal, or even in the lower cervical, region, but a few cases have been recorded in which it persisted and was even exaggerated. Reference has already been made to this subject. There is no constant change in incomplete transverse lesion above the lumbar enlargement.

The knee-jerks are always exaggerated if there is degeneration of the central motor neurones without degeneration of other parts of the spinal cord; and there may be actual clonus of the quadriceps muscle when the patellar tendon is struck, giving rise to a series of kicks of the leg, and these kicks, or at last the first one or two, may be so violent that the leg is extended ad maximum. Contractures of the adductors and occasionally of the quadriceps of the other leg are also nearly always present. Clonus of the patella usually co-exists and may be of a permanent character,—that is, continue as long as the pressure is maintained. This degeneration of the lateral columns occurs in the doubtful primary lateral sclerosis, and in cases of various cerebral lesions involving the motor tract. It occurs in the apparently idiopathic degenerative disease of the cord known as amyotrophic lateral sclerosis. In syringomyelia or in partial lesions of the dorsal or cervical cord arising from pressure as a result of disease of or of injury to the spinal column, the knee-jerk is usually increased, the increase ranging from a moderate exaggeration to (rarely) the extreme spastic type. In nearly all these cases there are associated sensory disturbances which aid in rendering the diagnosis clear. These may be paræsthesia, often in the form of a girdle sensation at the level of the lesion, dissociation of sensation, as a rule more pronounced at first in the foot, but often ascending; a partial or complete anæsthesia, the latter sometimes involving all forms of sensation and limited by a horizontal line extending around the trunk at the site of the lesion. A distinction may be drawn between the exaggeration of the patellar reflexes with and without paralysis. In cases of hemiplegia the knee-jerk is usually greatly increased on the paralyzed side, in 92 per cent. of the cases, and moderately increased on the sound side, the contractility of the muscle being not in the least affected by the absence of voluntary innervation. In the first few days the reflex may be diminished or lost. Strümpell has observed a diminution in old hemiplegias. In the syndrome to which the name primary spastic paraplegia was formerly given, the power of the muscles may be only slightly impaired, but their extreme spasticity renders it difficult for the patient to control them. In lesions of the upper portion of the cord the paralysis may be complete and yet the reflex be exaggerated. In functional nervous conditions, such as hysteria and neurasthenia, the knee-jerks are nearly always increased; although very rarely they may be absent and hypotonia be present. There is no clonus and the patellar jerk, as a rule, does not show nearly the same degree of exaggeration. The power and voluntary control of the muscles in these cases may be intact. In general diseases of the brain the knee-jerks are variably affected. In cases of disease of the cerebellum or frontal region they may be increased or decreased, and periods of increase and decrease may alternate in the same case. In cases of disease of the cerebral hemispheres associated with increased intracranial pressure, as, for example, tumor, hydrocephalus, or meningitis, the knee-jerks, as a rule, are increased. In cases of brain tumor, careful statistics indicate that they may be of some aid in local diagnosis, although their behavior is not constant. Müller, in fifty-five cases of tumor of the frontal lobes, found the patellar tendon reflex absent eleven times, normal nineteen times, and increased twelve times; in two cases it was lost on the side opposite to that of the tumor. Williamson found it absent in 20 per cent. of forty-five cases of lesion of the frontal lobes. It is apparently absent in about 3 per cent. of tumors in other parts of the cerebrum. Reh finds it diminished in 34 per cent. of all cases of brain tumors. In tumors of the cerebellum it is lost in 41 per cent. of the cases according to Voss, and in 29 per cent. of the cases, according to Dinkelburg. In a case that I observed it disappeared and reappeared twice. The abdominal reflex is usually preserved, but this appears to be of no value as a differential sign. In chronic encephalitis—that is, in general paresis—the knee-jerks are usually increased, but occasionally are diminished or absent, especially after flaccid apoplectic attacks. The cases in which this occurs are usually recorded as the tabetic form, and lesions of the posterior columns of the cord are found at autopsy. Sometimes they are unequal. In insanity there is no characteristic change in the knee-jerk. In epilepsy the knee-jerks are, as a rule, increased, particularly in those congenital epileptics whose brains show actual lesions. In shock they are usually lost, and for this reason are often absent immediately after an epileptic attack. In uræmia they may be increased (Fürstner). In certain general diseases the knee-jerks may be altered. In diabetes they are usually lost; in febrile conditions, especially typhoid and tuberculosis, they are increased. In croupous pneumonia it has been asserted and denied that they are diminished. When two conditions co-exist that affect the reflex in opposite ways, the result is variable. It has been observed to appear in the paralyzed limb in hemiplegia occurring in the course of tabes (Westphal). It is now generally admitted that the arc is completed in the third and fourth lumbar segments, with occasional variation to a segment above or below. There have not been wanting investigators, however, who believe that the arc is completed in segments at higher levels, or even in the brain, but little importance can be attached to their views. According to Sherrington, the sensory nerve in apes is the branch leading from the vastus internus muscle to the lumbar plexus. After section of all other sensory nerves, excepting this, the reflex can still be produced. The motor nerves are, of course, those passing to the different members of the quadriceps and adductor muscles on either side.

The Patellar Reflex.—This consists of a sharp contraction of the quadriceps muscle drawing up the patella when that bone is forced downward by a blow. In other words, it is a reaction of the quadriceps muscle to sudden extension. The reflex is best elicited if the leg is extended. The patella may either be grasped between the thumb and index-finger and pushed forcibly down, or the finger may be laid transversely across the upper border and struck a smart blow with the percussion-hammer; or some round object, such as a lead-pencil, may replace the finger, or the ingenious instrument devised by Mills, which consists essentially of a ring encircling the patella, the lower end provided with a handle and the upper end with a slight enlargement upon which the blow is struck. The patellar jerk is not nearly so constant as the knee-jerk. It occurs, however, in the majority of normal individuals. In cases of spasticity of the muscles of the thigh the jerk is violent and may be repeated in the form of a tonic spasm, known as patellar clonus. This reflex has the same significance as the knee-jerk, but may be tested with advantage in patients who, through disease, stupidity, or ignorance of the language, cannot or will not relax the leg sufficiently to obtain a satisfactory test of the knee-jerk. As clonus indicates disease of the pyramidal columns, it is of considerable clinical importance.

The adductor reflex consists of a sudden contraction of the adductor muscles on the inner side of the thigh when a blow is struck just above the internal condyle of the femur. There is usually a slight adduction of the thigh and occasionally a slight painful sensation. The reflex is best obtained if the thigh is flexed at about a right angle upon the trunk and the knee flexed upon the thigh. It is not invariably present in health, about 34 per cent., but is always present in spastic conditions, or, in fact, in any condition that gives rise to exaggeration of the knee-jerk. Reflexes may also be occasionally obtained by striking upon the *hamstring tendons*. For this purpose the patient should lie upon the face, the leg should be lifted from the bed by an assistant until the tendons of the biceps or of the semimembranosus and semitendinosus are relaxed, then the tendon to be tested should be moderately compressed by the thumb upon the dorsum of which a sharp blow is struck with a hammer. For some reason these reflexes have never excited the interest of clinicians and are rarely tested. It is not known whether they are present in all normal persons, but they appear to be subject to the same conditions of increase and decrease as are the other tendon reflexes of the leg.

The Achilles-tendon reflex consists of a sharp contraction of the muscles of the calf if a blow is struck upon the Achilles tendon in a state of moderate tension. To elicit this reflex the leg should be flexed upon the thigh at about right angles; the foot is then forcibly thrust upward until the tendon is tense, when it is struck a sharp blow. There is a contraction of the soleus and more or less extension of the foot. It seems to be generally admitted now that this reflex is produced only by the soleus muscle, and, in fact, is more easily obtained if the gastrocnemius is relaxed. Both the afferent and the efferent fibres pass through the more sensitive inner portion of the sole with some force. There is some dispute as to whether a sudden dorsal flexion of the great toe constitutes the Babinski reflex or not. As it appears that even this sudden movement occurs very rarely, if at all, in normal persons or in those suffering from functional nervous disease, it may be accepted that it is really a pathological manifestation. Although usually associated with exaggeration of the tendon reflexes, this is not invariably the case. The value of the Babinski reflex compared with other signs of disease of the pyramidal tracts in the cord has been a subject of considerable investigation. It appears to be more constant than ankle-clonus, and as constant as the increase of the knee-jerk. It is said not to occur if the thalami have been injured, but this lacks confirmation. In my experience, moreover, it appears to occur earlier and in certain cases to persist after the other signs have disappeared. In two cases of valvular heart disease which developed hemiplegia whilst under observation, probably embolic in nature, the Babinski reflex appeared during the first six hours, and before marked exaggeration of the other reflexes was apparent. In a case of hemiplegia with complete hemianæsthesia and abolition of all the tendon reflexes throughout the course of the disease, which lasted four months and terminated fatally, the Babinski reflex was occasionally obtained. In three cases of old hemiplegia with almost complete restoration of function and only slight increase in the knee-jerk and an imperfect clonus, the Babinski reflex was invariably obtained on the affected side. It has been claimed that it may occur in functional nervous diseases, especially hysteria, epilepsy, deep sleep, coma, and narcosis, and in typhoid fever. Münch-Petersen, whose work requires confirmation, claims to have obtained it in a considerable proportion of healthy adults. Cohn regards it as of little value. It often occurs in normal infants, and therefore is of no clinical value before the age of two years (Morse). It has been questioned whether a permanent dorsal extension of the great toe, such as sometimes occurs in Friedreich's ataxia or old hemiplegia, may not be a form of the Babinski phenomenon. Its arc is completed in the lower sacral region.

Modifications of the Babinski Phenomenon.—The discovery of the Babinski reflex stimulated great interest in the cutaneous reflexes in general, and particularly in those of the lower extremity. Oppenheim has obtained dorsal flexion of the toes in pyramidal disease by stroking the inner side of the leg, and his assistants have endeavored—although unsuccessfully—to give this the rank of an independent reflex. Schaefer has obtained the same reaction by pinching the Achilles tendon. Hirschberg produces slight adduction of the foot somewhat similar to the Babinski sign, by stroking the inner side of the instep. He claims that this reflex never occurs unless there is organic disease of the pyramidal columns, but his statement has been denied by Crocq. Mendel and von Bechterew have described a somewhat analogous reflex obtained by striking the dorsum of the foot. Under normal conditions there is dorsal flexion of the second to the fifth toes. In pyramidal disease plantar flexion of the same toes may occasionally occur.

The femoral reflex was described by Remak. It consists of a plantar flexion of the toes and extension of the foot and leg if the upper portion of the anterior surface of the thigh is irritated. It only occurs in severe lesions of the cord, such as transverse myelitis occurring above the eighth dorsal segment, the arc, according to Remak, being completed in the lower portion of the dorsal cord. It seems not unlikely, however, in view of the muscles involved, that the segments containing the arc are really lower than this.

Unclassified Reflexes.—Certain phenomena whose exact nature has not been determined, but which are akin to reflex action, may sometimes be observed.

Kernig's sign was first described in 1888 in that birthplace of many reflexes, St. Petersburg. It consists of a spasmodic contraction of the biceps, semimembranosus, and semitendinosus when, if the thigh is flexed at right angles to the trunk, an attempt is made to straighten the leg upon the thigh. There is some dispute as to the point at which Kernig's sign is said to occur, Kernig himself stating that the reflex was present if the leg could not be extended over an angle of 135° with the thigh. In its most typical form, however, the contraction takes place as soon as the angle of 90° is reached. This reflex may be elicited either by having the patient sit in a chair and then attempting to pull forward the leg, or, if he is lying on his back, flexing the thigh upon the trunk at right angles, and then attempting to extend the leg. One hand should be placed in the popliteal space and the sudden tension of the hamstring tendons is a very characteristic phenomenon. Kernig originally considered the sign pathognomonic of meningitis, in practically every form of which he supposed it occurred, and subsequent investigations apparently confirmed this opinion (Friis, Netter, Herrick). Of late, however, it has been found to occur in focal brain disease (Sailer) and in the course of certain infectious fevers (Shields), and it does not occur in all cases of meningitis, and in particular it is likely to be absent in tubercular meningitis (Dieulafoy). It is, however, still a valuable sign because, although not pathognomonic, it occurs in more than 90 per cent. of all cases of acute meningitis, and very rarely in other conditions. Therefore, if found, the probabilities are strongly in favor of the existence of meningitis.

A reflex whose nature is not clearly understood is **Sinkler's toe-jerk.** It consists of forcible flexion at the hip and leg when the great toe is flexed strongly on the sole. It appears to occur in states of extreme spasticity, and I have been able to elicit it several times in cases of transverse myelitis or spastic paraplegia. Nothing appears to be known concerning its mechanism, but the arc must be completed in the lumbar portion of the cord, and it is probably one of those vestigial reflexes, only developing when inhibition is entirely removed, of which Strümpell speaks. The excitation appears to be due to traction upon the extensor muscles of the foot.

Front-tap was first described by Gowers. It is elicited as follows: The leg is flexed slightly dorsally by the observer, and a blow is struck upon the tibialis anticus muscle. In about 40 per cent. of normal persons there is plantar flexion of the toes. The reflex appears to be more common in various forms of functional nervous disease, hysteria, neurasthenia, etc., and to be absent invariably in hypotonic conditions, such as tabes dorsalis.

The **tibialis phenomenon** (Strümpell) consists of the contraction of the tibialis anticus muscle when the thigh is drawn towards the body. As the patient lies upon the back, one hand is placed over the knee and the other over the dorsum of the foot. The knee is then lifted by the patient when, if the reflex is present, the tense tendon of the tibialis anticus muscle is felt by the hand over the foot. This reflex occurs in cases of spastic hemiparesis, and may be of some practical value.

REFLEXES OF THE TRUNK.

Apparently there are no tendon-reflexes connected with the trunk. Two important skin-reflexes have been described, the abdominal and the cremasteric. The **abdominal reflex** consists of a sharp contraction of the muscles on one side of the abdominal wall when a light stroke is made from that side, passing obliquely downward and outward. This reflex is produced by irritation of the cutaneous terminations of the intercostal nerves that continue downward from the intercostal spaces and are distributed to the skin of the abdomen. The afferent and efferent fibres pass through the intercostal nerve trunks, and the reflex is therefore completed in the dorsal region of the spinal cord. It is customary to recognize two parts: the reflex of the upper and of the lower quadrants, or the upper and lower abdominal reflexes. There is no actual anatomical or physiological dividing line. This reflex is present in 98 per cent. of normal persons, but varies very considerably in different persons or in the same person at different times. It is increased by the conditions that increase the skin reflexes; it is abolished by lesions of the spinal cord which destroy the dorsal region. Sometimes if only one part and not the other is involved it serves, in a sense, as an aid to localization. It is often active in tabes dorsalis, and may be present in cases of hysterical anæsthesia, but is lost in cases of organic anæsthesia of the abdomen. The **hypogastric reflex**, recently described by von Bechterew, consists of a deepening of the suprainguinal region when the skin of the inner upper part of the thigh is irritated. According to Crocq it does not occur exclusively in women as was at first believed.

The cremasteric reflex consists in the upward jerking of the testicle when the skin on the inner surface of the thigh is irritated. It is most readily elicited by pinching, scraping, or pricking the skin over the adductor muscles about one inch below Poupart's ligament. The upward movement of the testicle is sudden and independent of any contraction of the scrotum, but may be associated with twitching of the abdominal muscles. The contractility of the cremasteric muscle varies extraordinarily in different individuals. A small proportion—estimated at 1 or 2 per cent.—have voluntary control over the muscle and can elevate the testicle at will, and in these cases the reflex is pronounced to the degree of exaggeration. In other individuals without obvious lesion of the central or peripheral nervous systems the reflex may be persistently absent. In other cases it may be extremely variable. Under pathological conditions, it is increased in functional nervous states; it is usually increased in conditions giving rise to increased spasticity, although it may be diminished in hemiplegia on the affected side, and is lost in conditions causing hypotonicity, as, for example, tabes dorsalis. It is said to be exaggerated in sciatica. It is a reflex of moderate clinical importance. The contraction of the dartos muscle lining the wall of the scrotum is occasionally mistaken for the cremasteric reflex. It is, however, independent and differs distinctly in character, being a slow contraction coming on at an appreciable interval after the skin has been irritated. It persists for a short time, and then relaxation ensues. This reflex appears to be independent of nearly everything excepting complete organic anæsthesia of the part irritated. It is of no clinical importance.

Von Bechterew has described a **pyramidalis reflex**, a slight contraction in the lower part of the abdomen just above the symphysis when the skin above this region is irritated.

The periosteal and muscle reflexes are not important.

Scapulohumeral Reflexes.—Von Bechterew has described a series of reflexes connected with the scapula, which are best elicited by striking on the spinal edge just below the superior internal angle. This causes a slight contraction of the infraspinatus and occasionally of the supraspinatus muscle, and adduction and external rotation of the humerus. In cases of hemiplegia in which the muscles are spastic the contractions may be more extensive, involving also the trapezius and deltoid, and causing, therefore, abduction instead of adduction of the arm. Not infrequently I have observed a contraction of the muscles of the opposite shoulder. It would seem, therefore, that the scapular reflex must be a true reflex and not merely the result of muscular irritation. There is no doubt, however, that striking upon the bellies of the spinati muscles gives rise to contraction of these muscles with results that simulate the reflex under ordinary conditions. Steinhausen regards it as a periosteal reflex constant in normal persons.

Iliac Reflexes.—Striking upon the crest of the ilium gives rise to various contractures of the muscles attached to the pelvis. These reflexes are increased by all conditions giving rise to increase of the muscular irritability. They have received very little attention from neurologists, and little is known excepting the fact of their existence.

Negro has described as the **gluteal reflex** a sharp tetanic contraction of the gluteal muscles when pressure is made over the sciatic foramen in cases of sciatica. It may assist the diagnosis in doubtful cases.

Mention may be made of the idiopathic muscular contraction, or *myoidema.* This consists of a tonic spasm of isolated fibres of a muscle which have been subjected to vigorous mechanical irritation. It is best elicited in the pectoral muscles by tapping sharply upon them with the finger or hammer. This produces a small lump which rapidly disappears. It can also be elicited in the biceps by pinching it sharply, a ridge appearing along the line of the pinch. This reaction, although it can usually be obtained in normal persons who are thin and whose muscles therefore are not protected by a layer of fat, is most striking in cachectic conditions. It also is common in certain forms of insanity, as general paresis, dementia præcox; in epilepsy, cerebral syphilis, etc. It has no clinical significance, excepting as a confirmation of cachexia, which is, of course, readily recognizable by other means.

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GAITS.

THE GAIT AS A SYMPTOM OF NERVOUS DISEASE.

Walking may be described as progression by placing one foot on the ground, supporting the body upon it, lifting the other foot to a slightly higher level, and swinging the leg forward; this leg in turn becomes the support, and the first leg repeats the movements. The supporting leg is kept rigid by the contraction of the muscles, particularly those of the calf, the quadriceps, and the gluteal and psoas muscles, although practically all the other muscles of the limb are brought into play for the purpose of limiting movements at the joints. During the act of walking there are two chief movements in the supporting limb: at the ankle-joint, in which the tibia rolls forward on the astragalus, and at the hip-joint, in which the thigh-bone rocks backward in the acetabulum as the body moves forward. There is also a slight rotary movement in the hip as the pelvis is swung around it in the effort of throwing forward the opposite leg. The knee-joint usually remains fixed until the movement forward becomes so great that the heel of the supporting leg is lifted from the ground, when it begins to bend slightly. The shortening of the swinging leg is accomplished by tilting the pelvis and slight flexion at the thigh, knee, and ankle. The swing commences with a slight throw given with the toes as the foot leaves the ground, and terminates as the heel strikes the ground, when the forward inclination of the supporting leg becomes so great that the swinging leg no longer clears the surface.

METHODS OF STUDY.

The ordinary mode of walking may be modified by structural deformities, especially if in the skeleton, or by functional defects either in the muscles or in the nervous mechanism controlling them. These modifications have been studied usually by simple observation, but a variety of methods have been employed for the purpose of elucidating the final changes and making permanent records. The simplest method is to measure off a definite path on which the patient walks. The number of steps is counted and divided into the distance travelled. The length of each step is thus readily determined. By observing the patient carefully from behind or in front, lateral deviations in the movements of the legs can be detected, and by placing the eye close to the ground it can be noted whether the feet are abnormally raised or not. This method, however, is scarcely better than direct observation (Weber).

It is a distinct advance to employ some method by which the footprints themselves can be more or less permanently impressed upon the surface. This is, of course, done upon soft soil or sand, but the yielding character of the material produces certain alterations in the gait which render it slightly abnormal. Moreover, the preservation even of a single footprint by the method of making plaster casts is tedious and difficult.

Vierordt was apparently the first to employ a color material, which was exuded by a specially prepared shoe. Gilles de la Tourette covered the sole of the foot with powdered oxide of iron, and then had the patient walk upon strips of rough paper, enough of the powder being caught in the interstices to leave a record. Still more complicated is the method of Mönkenmöller and Kaplan, who employ an alcoholic solution of chloride of iron in which the stockings are soaked. The patient walks upon a piece of paper which is afterwards sprayed with an alcoholic ethereal solution of ammonium sulphocyanide, forming ferric sulphocyanide, and leaving a permanent dark red mark that can readily be photographed. The very thick wet stockings interfere slightly with the gait.

I have used a simple modification which has proved so satisfactory that I believe I can commend it heartily. It consists in painting the feet with ordinary black paint,—that is, lamp-black dissolved in linseed oil. The paint should be somewhat thicker than that ordinarily supplied ready-made. It can best be smeared on the feet with a wad of cotton or gauze. Ordinary wrapping-paper is employed. This comes in rolls about four feet wide, from which strips of convenient length (usually from fifteen to twenty feet) can readily be cut. A single painting suffices for about twenty clear tracings from each foot,—that is, the patient can walk a much longer distance than the usual length of the strip of wrapping-paper. The paint dries quickly and makes an impression that is practically permanent. I have records over four years old that are as clear as they were originally. The strips can be rolled up into very small compass, can easily be stored and classified, and photographic reproduction is very satisfactory. Jendrassik condemns these methods because, according to him, it gives us no more information than simple inspection. In this I think he is mistaken. Obviously it enables us to substitute for more or less erratic guessing actual measurements regarding the length of the steps, the difference in the length of the steps in the two legs, the separation of the legs, and whether or not successive steps of the same leg are in a straight line. Moreover, the print of the foot can be carefully studied: whether the whole of the sole touches the ground, whether part of it remains permanently above the ground, and if any dragging occurs it is very distinctly shown. The great advantage of this method, however, is that it furnishes a permanent record infinitely preferable to any description that can be written. The chief objection to it is that no record is made of the movements of the feet while in the air.

A very simple method which may be employed if no materials are at hand is to allow the patient to walk on the floor after the feet have been wet. Usually from five to ten steps with each foot will be recorded, and the water does not evaporate for several minutes. If the patient wears thick stockings which have been saturated with water a greater number of steps can be obtained. This will enable the observer to take satisfactory measurements of the foot-prints.

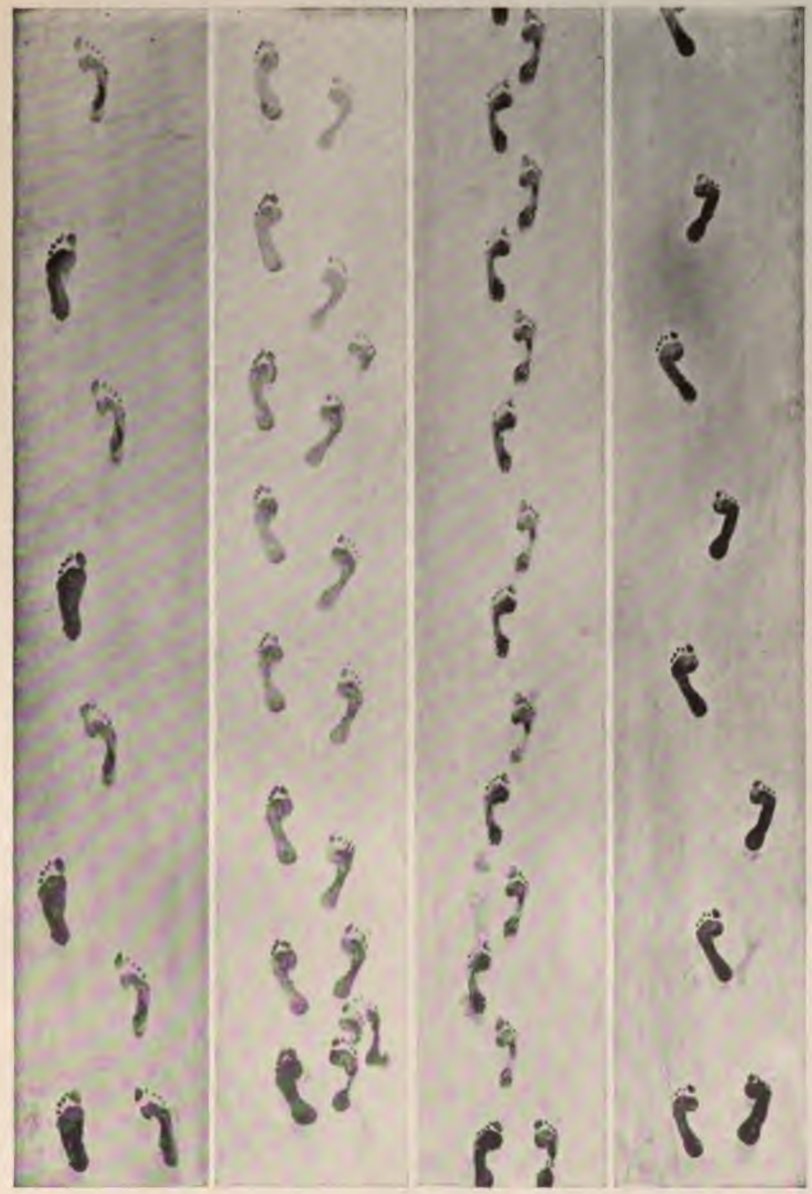
The application of instantaneous photography to moving objects provided a new method for the study of the gait. To Muybridge, working at first independently and subsequently under the auspices of the University of Pennsylvania, the credit for the first complete study of the human gait is due. Not only can the individual phases of motion be studied by this method, but also by various methods the pictures can be thrown upon a screen in such rapid succession that the movement appears to be reproduced. For scientific purposes it suffers under the disadvantage that pictures taken with the subject advancing directly towards or retreating from the camera are comparatively unsatisfactory. This method, with various changes, has also been employed by Marie, Dercum, Braune, Fischer, and Londe: and Marinesco has used the more modern cinemetograph.

Jendrassik has devised a modification which he considers superior to all other methods. It consists in having the patient walk in a dark room in front of a photographic plate and lens, and then by means of electricity producing momentary flashes of light at regular intervals. As a result the picture of the individual at various stages of the gait appears upon the plate. The objections to this method are numerous. In the first place, the subject walks in a room alternately dark and bright, and as a result the gait must be to a certain extent disturbed. In the second place, and to my mind this is a more serious objection, the pictures upon the plate are superimposed; it is very difficult, therefore, to make a careful analysis. In the third place, it is practically impossible to take pictures with the patient walking toward or away from the camera. Nevertheless, this method, if carefully carried out, yields excellent results, and some of the pictures published by Jendrassik leave little to be desired.

Other methods that have been employed for attempting to record various movements graphically, either upon a rotating drum or by means of an apparatus squirting colored fluids upon a recording surface, are so cumbersome and unsatisfactory that they have been entirely discarded.

The *classification* of the pathological varieties of gait is comparatively simple theoretically. Practically, however, it is difficult, partly because of the great variation occurring in many of the forms, partly because of the not infrequent association of two or more disturbing factors. Broadly we may recognize two groups: those due to actual deformity of the skeleton and those due to disturbances in the neuromuscular mechanism. The former, with the exception of a few forms of trophic disturbance of the bones, belong to the domain of surgery, and will not be considered in this article. The latter may be divided into five groups. First, flaccid paralytic lesions of the neuromuscular mechanism; second, spastic paralytic lesions of the neuromuscular mechanism, each of these may be subdivided into the unilateral and bilateral forms, and they are often combined; third, the group characterized by loss of muscular co-ordination,—that is, the ataxic gaits; and, fourth, a group due to involuntary muscular movements,—the chorei-

PLATE D.



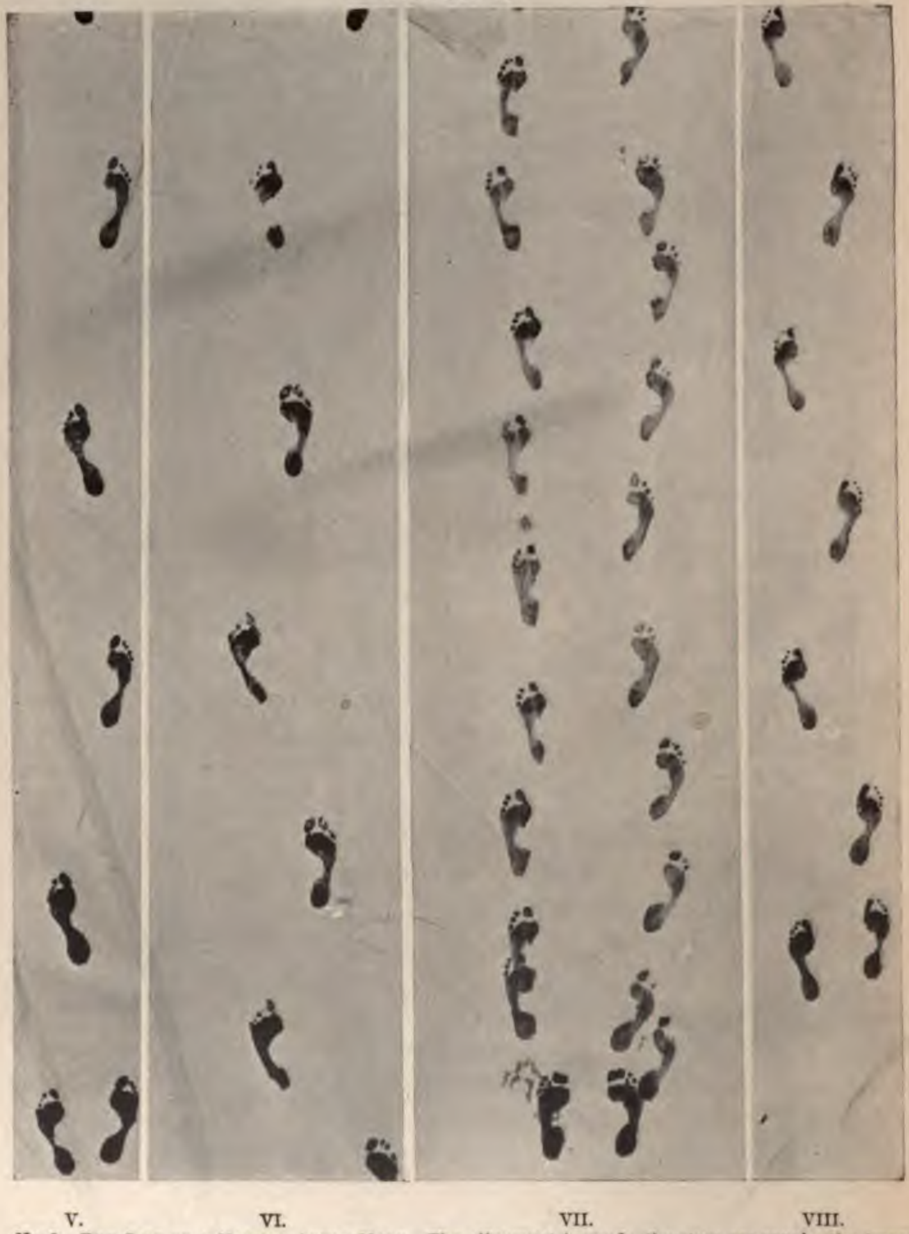
I. Case of Hemiplegia, Two Years Duration.—The impression of the right foot is weak: there is some contraction of the muscles, causing accentuation of the arch. The alignment is good and there is very little dragging of the foot.

II. Case of Hemiplegia of Long Duration.—The right foot is turned out and drags distinctly (unfortunately the illustration has been retouched and does not show this). The steps are uneven and there is occasional staggering.

III. Case of Cerebral Spastic Paraparesis.—The steps are short and uneven and there is a tendency to crossing the legs.

IV. Case of Primary Neural Atrophy; that is, Paralytic Paraparesis.—The toes are turned out; the alignment is poor. The peculiar character of the footprint is due to the atrophy of the muscles of the sole of the foot.

PLATE E.



No. 5. THE GAIT OF A NORMAL ADULT MALE.—The alignment is good; the steps are equal; the impressions are clear and distinct. The left foot is turned out slightly more than normal.

No. 6. A CASE OF TABES DORSALIS —The alignment is poor; the distance between the steps is unequal; at one point the leg is thrown slightly to the side. The impressions vary in intensity, particularly with reference to the arch of the left foot.

No. 7. A CASE SUPPOSED TO BE CEREBELLAR DISEASE.—The type of gait is the late stage of ataxia with paresis. The steps are very short; the legs are widely separated; the alignment is poor. The steps are very unequal in length, and the impressions vary in intensity. It will be noted that the number of toes striking the ground varies in different impressions, showing a slightly spastic condition of the extensor muscles.

No. 8. THE GAIT OF A NORMAL ADULT FEMALE. The steps are shorter, and the separation of the legs slightly greater than in the tracing taken from the adult male. The gait presents no abnormal features.

form gaits. This comprises a number of very diverse conditions. Finally, there are several forms, different in themselves and not belonging to any of the regular types. Before discussing these different classes I wish to mention the general features that should be most carefully studied, because it is by means of the amount and degree of their deviation from the normal that the character of the gait is determined.

First, the length of the steps; whether the step made by one foot is longer or shorter than the step made by the other,—that is to say, whether the distance from the toe of the left foot to the heel of the right foot is more or less than the distance from the toe of the right foot to the heel of the left foot. Second, the degree of separation of the two feet; under normal circumstances, a straight line drawn between the footprints should touch or slightly cut the inner side of both heels. Third, the degree to which the toes are turned out; this is modified more or less by habit, but normally a straight line drawn through the centre of the heel passes through the ball of the great toe; however, moderate deviations from this rule cannot be considered pathological. Fourth, the part of the foot which first touches the ground, whether the heel or toe; this is also modified by habit, but it is excessively rare for any person normally to strike the ground with the ball of the foot first, although Ellis believes that certain savage races normally walk in this manner. Fifth, the way in which the foot leaves the ground; whether it is thrown clear by the spring of the toes or whether they remain in contact with the surface for a longer or shorter time while the foot is dragged forward. Sixth, the way in which the sole of the foot rests upon the ground, in some cases the arch is distinctly lost, or the patient may walk upon the toes or the side of the foot. Seventh, the height to which the foot is lifted from the ground during the step. Eighth, the amount of lateral swing in the leg. Ninth, the manner in which the foot strikes the ground, whether forcibly or lightly. Tenth, the alignment of the steps. In addition, such gross changes as cross-legged progression or walking with crutches should be noted, and such finer changes as the behavior of the toes during the step. Under certain circumstances, spastic dorsal flexion may prevent one or all of the toes from touching the ground at any point.

THE PARALYTIC GAITS.

The flaccid paralytic gaits are characterized by the absence of the reflexes in addition to the weakness of the limbs. As a general rule, these gaits occur in various forms of peripheral palsies and myopathies. If bilateral, such, for example, as occurs in **peripheral polyneuritis**, the gait presents the following peculiarities: the steps are short, the feet are planted rather widely apart, and there may or may not be irregularity in alignment. The feet are lifted from the ground without swinging, the toe leaving last. The knee is lifted somewhat higher than normal from the ground, in order to permit the toe, which, as a result of toe-drop, is not raised, to clear the floor. The toe strikes the ground first and then the rest of the foot is planted upon the surface. During the step the foot remains flat upon the ground and does not aid in lifting the body. Walking is therefore accomplished chiefly by the muscles that move the thigh upon the pelvis. In the severer forms of this type of paralysis the weakness is so great that it is impossible for the patient to lift the feet from the ground, and they are dragged along, usually turning slightly outward. In these forms additional support is nearly always required, either by canes or crutches. As a rule, walking is attempted only in the milder forms.

In **anterior poliomyelitis,** either unilateral or bilateral, the type of gait depends largely upon the particular muscles affected. If the involvement is extensive, walking without crutches is impossible. The legs are drawn forward and serve as a sort of third crutch to aid in progression. Very often it is necessary to stiffen the joints by means of braces. If the peroneal muscles are involved there will be foot-drop, the leg will be lifted higher from the ground, and will be planted nearly flat, the toe touching first, but if the calf muscles are preserved there will be a distinct spring at the end of the step. In these cases the foot is usually turned out. If contractures have ensued, giving rise to club-foot, the condition becomes surgical rather than neurological, and the type of gait depends upon the nature of the deformity.

In the ordinary forms of **muscular dystrophy** the ability to walk is preserved for a long time. The gait is quite characteristic. The steps are short, usually made with effort; the legs are well separated so that the gait has a waddling character; the feet are not lifted very well from the ground, and the knees are kept nearly completely extended. If at any time they become flexed the patient is likely to fall. The foot, however, usually clears the ground, and, unless the weakness is extreme, the alignment is good. As the muscles of the trunk are also frequently involved there may be considerable rocking of the pelvis which accentuates the waddling.

In *pseudomuscular hypertrophy* the gait is essentially the same, the only difference being in the appearance of the limbs. In both forms the patient shows a peculiar method of arising from the floor which has been frequently described, but is so characteristic that it serves as an important symptom. He first assumes a sitting posture, then drawing the body forward gets upon the hands and knees, then pushing the body backward he brings the hands close to the feet, grasps the legs, and gradually climbs up his own legs, supporting the trunk partly upon the arms and partly upon the legs. In the peroneal form of muscular dystrophy and in some forms of polyneuritis the predominant characteristic of the gait is the extreme ataxia. This will be described in connection with ataxic gaits.

The Spastic Paralytic Gait.—**Lateral Sclerosis.**—Bilateral paretic disturbance of gait with spasticity of the muscles occurs as a result of cord or brain lesion. Pressure upon the cord above the lumbar enlargement gives rise to a peculiar so-called spastic gait which was formerly believed to be due to sclerosis of the lateral columns. As a matter of fact, a descending sclerosis of these columns does occur, but very rarely as an idiopathic lesion. The steps are equal in length and may be short or long, according to the degree of the paralysis: the less the paralysis the longer the step. The feet are usually kept moderately close togther, often, indeed, there is a more or less pronounced tendency for the legs to cross. During the swing the leg rotates slightly outward to pass the other leg; the leg is kept stiff and the foot extended. As a result, for a greater or less portion of the stride the toe remains in contact with the ground. The whole gait is of a peculiar stiff and awkward character. Although the toe touches the ground first, there is no spring to it, and the patient proceeds very much as if walking on stilts. It is characteristic of this condition that the toes of the shoes are very rapidly worn out, and that frequently the inside of the legs of the trousers, particularly at the knees, are also soon worn through. In forms in which the spasticity is the most pronounced condition the legs may even be crossed, giving rise to cross-legged progression. A modification of this form occurs in *Little's disease*. In this, however, there is marked foot-drop; the patient therefore lifts the leg high from the ground in order to clear the toe: the so-called "chicken-gait."

The unilateral spastic paretic gait is, in the vast majority of conditions, the result of cerebral lesion. The gaits of **hemiplegia** are, as a rule, quite characteristic. The degree of modification, of course, depends largely upon the extent of the paralysis. In the mildest forms there is merely a slight limp. Careful observation and foot-print records, however, will show that even in this type the step on the hemiplegic side is somewhat shorter; that the toe drags for an instant after the step has commenced, and that, as a result of the stiffness of the leg, it is swung slightly to the side, the foot being lifted less than the leg on the sound side. The steps are usually in a straight line; the foot is turned out, and not infrequently the feet are separated slightly more than in normal walking. The more severe forms are merely exaggerations of these features, but of course present a very different appearance. The step is short, the feet are well separated, the toe clings to the ground for a considerable distance, sometimes throughout the entire period of the step, the leg is swung well outward, and usually the footsteps on the affected side are no longer in a straight line, showing the existence of a greater or less amount of ataxia. As the muscles on the paralyzed side are not capable of tilting the pelvis, this is accomplished by throwing the body over to the opposite side, so that with each step it oscillates distinctly. The step on the sound side is usually characterized by an exaggerated spring from the toe, and a marked hurrying of the swing so that as little time as possible is spent resting on the lame leg. If the condition becomes more severe the patient is obliged to support himself with a cane or crutch usually carried on the sound side, although if the arm has recovered sufficiently it is not uncommon for the patient to carry a crutch on the sound side and a cane in the other hand.

GAITS DUE TO DISTURBANCE OF CO-ORDINATION.

The ataxic gait is characterized by the following features: inequality in the length of the steps; defective alignment of successive steps; irregular high lifting of the feet from the ground, and usually a somewhat stamping type of progression,—that is, the feet strike the ground rather vigorously. As a result of the uncertainty and irregularity of the footsteps, the centre of equilibrium is continually disturbed, and the body is jerked forward and backward or to one side or the other in order to preserve the balance.

In **tabes dorsalis** this type of gait occurs. All grades may be observed, from a slight impairment, which may be imperceptible unless special methods are used, to such a profound paresis and incoördination that the patient can only progress by the aid of two canes or crutches.

The tracing of a tabetic gait is usually quite characteristic. The spaces between the successive footprints show a considerable amount of variation. Occasionally a footprint is seen far to one side, due to the effort of the patient to recover his balance. Even where the walking is more regular, it can be seen that sometimes the feet are slightly crossed, sometimes more widely separated than normally. The character of the impression varies considerably. Sometimes the sole of the foot is pressed firmly against the ground; sometimes the patient walks upon the inner and sometimes upon the outer side of the foot. In some impressions the arch is distinct; in others it is faulty, apparently the result of irregular contractions of the muscles controlling it. The changes are always more pronounced the further the patient has walked. In the beginning the footsteps maintain a fairly good alignment, possibly due to the greater concentration of the mind in the beginning of the effort. The toes are sometimes turned out; sometimes the foot is directed forward, showing irregularities in the rotation of the leg at the hip. Even slight variations in the intensity of the impression can be made out in most of the tracings. One of the most characteristic features of the ataxic gait is that, in spite of the obvious impairment of equilibrium, only occasionally is an effort made to compensate it by separating the legs and thus broadening the base. Moreover, a study of the tracings shows that, in spite of the apparently long stride due to the elevation of the foot from the ground, and the forcible way in which it is placed downward, the average distance between the steps is slightly less than in normal persons of the same size.

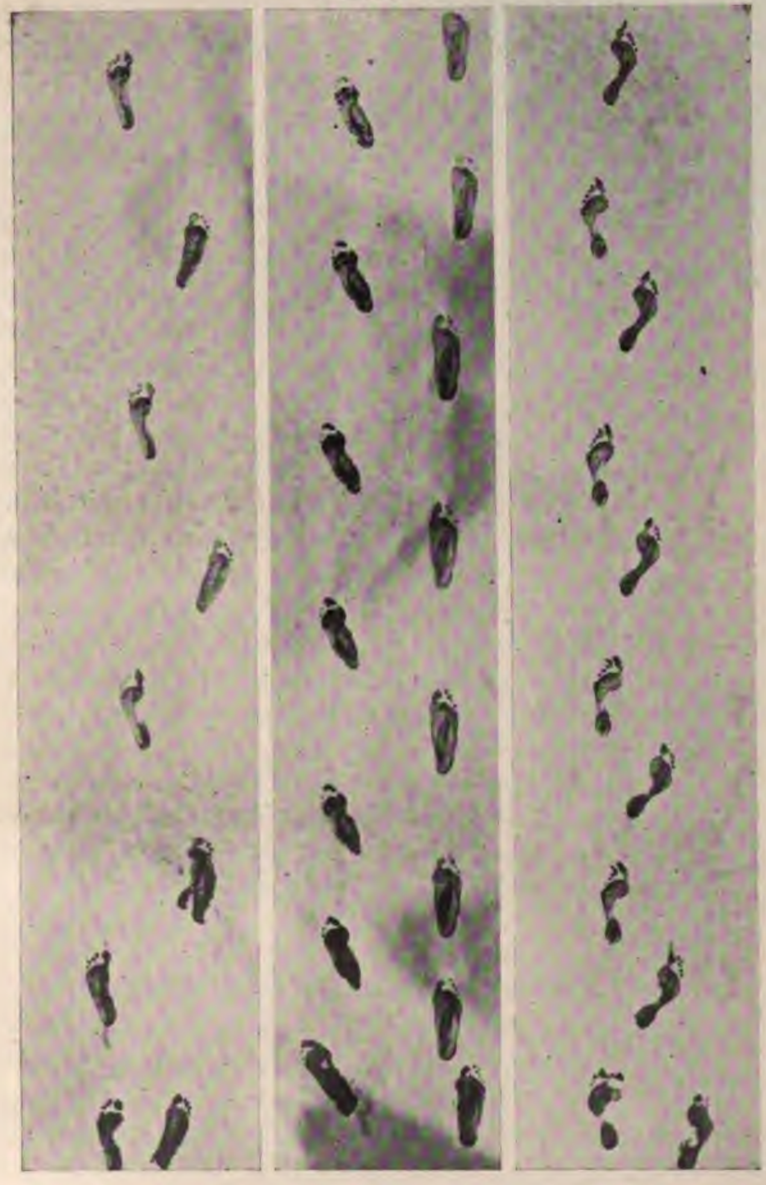
The ataxic usually appears to walk hastily and heedlessly, but it will be observed that he keeps his eyes fixed carefully upon the ground, and, as far as he may, chooses his path. There is also a certain element of tenseness and rigidity in the whole poise that is merely an indication of the effort made. In nearly any form of ataxia a footprint tracing will exhibit the characteristic features, even although they are not perceptible to simple inspection. But in these cases the alteration may be made more obvious by employing certain modifications in the gait. The simplest of these is to have the patient walk with the eyes closed. Ordinarily, as soon as the aid of vision is removed, the ataxia becomes much more pronounced. Walking backwards is accomplished with difficulty, and often results in falling. Walking upon an uneven surface is done very badly. For this reason such patients are very apt to fall over slight obstacles, particularly if entering a dimly lighted, unfamiliar room. They also walk badly in the twilight and at night. Indeed, it is in a measure a characteristic symptom of locomotor ataxia that the patients stagger in the early evening. Certain modifications occur in the gait as the result of the progress of the disease. Tabetic arthropathies produce such a relaxation of the ligaments of the joint that it may bend freely in all directions. Under these circumstances, with each step the leg shortens as a result of bowing, either backward or laterally. The body therefore tilts to that side, and the other leg can only be swung or dragged a short distance forward until it is necessary to change the step. Walking becomes difficult, but the deformity may become extreme before it becomes impossible, even although both knees may be affected. Arthropathies may occur in the hip, but do not give rise to such characteristic alteration in the gait.

In the advanced forms of *tabes with marked paresis* unaided locomotion is impossible. The patient usually seeks the aid of one or, often, two canes; the body is tilted forward, a good deal of the weight resting upon the arms, the steps are short, even dragging, and quite uncertain; the patient keeps his eyes fixed upon the ground as well for the purpose of placing the canes as of planting the feet. Even the movements of the arms are uncertain, and the canes are not carried forward in a straight line, but waver from side to side and are thrust against the ground in a manner that resembles the stamping of the feet. In cases of blindness as a result of optic atrophy in the course of the disease a curious modification takes place. The ataxia disappears almost entirely, and the patient may for a time be able to walk quite as well as any other blind person. As a rule, however, increasing paresis finally causes disability. As a result of careful education of the tabetic by means of systematic co-ordinated movements, gradually increasing in complexity (Fraenkel's method), so much improvement may ensue that even a severe ataxia may be converted into a mild form. In the *tabetic form* of general paresis the gait is modified in the same manner as in tabes dorsalis.

The gait in **Friedreich's ataxia** is intermediate between the spinal and the pure cerebellar forms, although it resembles the spinal more nearly. As the disease progresses the inco-ordination becomes more and more severe until finally the patient is unable to walk at all. Not infrequently a deformity of the feet—a form of talipes equinus—adds a limp to the gait of inco-ordination.

Cerebellar ataxia differs from spinal ataxia in several particulars. The co-ordination is usually more disturbed, therefore the steps are exceedingly irregular and lack alignment. Co-ordination is disturbed not only in the legs, but also in the trunk, and, as a result, there is considerably more swaying of the body, partly due to the inco-ordination, partly to the violent efforts of the patient to maintain equilibrium. The gait is not made worse by closing the eyes,—that is to say, visual impressions aid the patient little if any. There may or may not be a marked sense of vertigo. If the cerebellar type is pronounced the patient frequently falls, and, of course, in the severer forms walking unaided is impossible. It is noteworthy that in the severe forms crutches or canes are not of as great assistance as they are in spinal ataxia, and the patient must be supported by one or even two attendants.

PLATE F.



IX. X. XI.

No. 9. A Spastic Paretic Gait from a Case of Syringomyelia.—The paresis is in excess; the arch of the right foot is wholly lost, and that of the left foot is imperfect. The alignment is distinctly poor; the length of the steps is unequal, and the toes are turned slightly out. It will be noted that the intensity of the impressions is unequal.

No. 10. A Case of Left Hemiplegia of Six Months Standing.—The arch of the foot is wholly lost on the right side, and is imperfect on the left side. These changes possibly antedate the lesion. The foot on the paralysed side is turned outward at an angle of 30 degrees. The feet are well separated; the alignment is poor; the steps are very short. There is no distinct dragging of the toes.

No. 11. A Bilateral Spastic Gait.—The steps are short; the alignment is poor. The separation of the feet is unequal, but occasionally there is a tendency to crossing the legs. There is distinct dragging of the toes on both sides, the dragging of the foot being outward so that the knee can clear the knee upon the opposite side.

Cerebral ataxia is not very common. It occurs occasionally as a result of focal lesion, and is usually on one side of the body. Under these circumstances, if slight, it may be detected in the footprint tracing by noting that on one side the steps are slightly unequal in length and the alignment is poor. Bilateral ataxia may occur as a result of pressure from brain tumors or other conditions. It is usually of the cerebellar type.

In **multiple neuritis** marked ataxia sometimes occurs constituting the pseudotabetic form. This is especially characteristic in the hypertrophic form.

Choreiform Gaits.—The gait of **athetosis** is characterized by irregular movements not only of the legs, but also of the whole body. The muscles appear to relax and contract slowly, entirely independent of voluntary control, but at the same time a certain amount of control is possible, so that these irregular arhythmical movements can be partly inhibited and directed. By the exercise of great effort this inhibition and direction nearly always suffices to maintain the equilibrium. The patient walks with most irregular movements. The steps are unequal, the feet are planted irregularly, the body bends from side to side or forward and backward, the head is continually moving, and the patient appears as if falling backward or sideways and to recover his balance almost by a miracle. Actual falling rarely occurs. This condition is not incompatible with a fair degree of intelligence.

In ordinary **chorea** the gait is not typical. There is some jerkiness in the movements and occasionally paresis of the legs or of one side of the body, causing disturbances that resemble slightly those of hemiplegia.

THE GAITS OF FUNCTIONAL NERVOUS DISEASE.

In **hysteria** the greatest variety of disturbance of gait may occur. All the organic forms may be simulated more or less perfectly, and, in addition, there are certain types that are peculiar to hysteria. The commonest is the so-called histrionic gait. This can best be described as a series of attitudes during progression, with steps of an exaggerated, but not particularly abnormal, character. The body is usually held at various angles, the legs are drawn forward with a sweeping motion, the feet are lifted high from the ground, and there is considerable oscillation of the trunk. In these cases there is not really a true ataxia, although the footsteps may be somewhat uneven. In hysterical manifestations the symptoms are usually more pronounced when the patient knows he is under observation, but this is not invariably the case.

at this slow rate of progression voluntary stoppage is frequently impossible. In other cases, instead of becoming shorter as walking continues, the steps become distinctly longer, so that the patient is soon running with long strides. In taking footprint tracings of these cases I found one in which there was a marked dorsal extension of the toes when the sole of the foot was on the ground, so that the balls of the toes never came into contact with the ground, the patient touching with the heels and springing with the ball of the foot. As a matter of fact, the spring was insignificant, the foot being lifted and held almost horizontal to the surface. These patients also show retro- and propulsion,—that is to say, when standing or sitting they have a tendency from time to time to move or fall backward or forward. Patients with paralysis agitans, however, rarely fall, and, considering the difficulty in locomotion, it is remarkable with what readiness they undertake to walk either voluntarily or at the request of the observing physician. The nature of this gait has been the subject of some speculation, and it is supposed that the muscular rigidity characteristic of this disease does not enable the patient to recover the equilibrium after it has been slightly advanced in the course of movement. The station in these cases is quite typical. The patient bends slightly forward, the knees are slightly bent, the head slightly protruded, the neck is rigid, giving rise to a curious attitude, the elbows are thrown slightly back, the forearms are bent, and the hands slightly advanced. The whole attitude expresses an extreme degree of rigidity.

The gait in *sciatica* is, as a rule, characteristic. The patient walks with the leg flexed slightly upon the thigh, giving rise to shortening and a distinct limp. The foot is usually kept nearly horizontal to the ground, and there is a little spring from the toe at the end of the step. The swing of the leg is shortened and the patient, in order to put as little weight as possible on the affected side, throws the trunk toward the other. He also stands in a somewhat characteristic attitude. The leg is slightly flexed, the heel sometimes a little drawn up from the ground, and the trunk inclined toward the sound side.

DELAYED WALKING.

The age at which a child learns to walk is of some significance. As a rule, a normal child should stand alone before the first year and should walk before eighteen months old. This, however, is subject to many exceptions in both directions. Many children, especially European children, who appear to develop somewhat earlier than American children, walk before they are a year old, and a child may be perfectly normal in all other respects who does not walk until two years old, or even later. It is always advisable, however, to make a careful examination of any child who does not walk at eighteen months. Interruption in the ability to walk is of great clinical significance. It is not uncommon for children to walk a few steps at an early age, and then, as a result of fright from a fall, or of timidity, or of other unimportant causes, to refuse to attempt it again for several weeks. If, however, a child who has walked for any considerable time gradually loses the power, it usually means that some disease of the central nervous system is in progress. It is very common, for example, for children suffering from the various forms of degenerative atrophy, particularly of the hereditary types, to lose the power of walking at about the time these diseases manifest themselves. Thus, in cases of the amaurotic form of family idiocy the children cease to walk about the age of two years. This is also true of many other forms of idiocy.

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CHAPTER XXIII.

DEGENERACY (DEGENERATION, CONSTITUTIONAL DEFECTS, DEVIATION.)

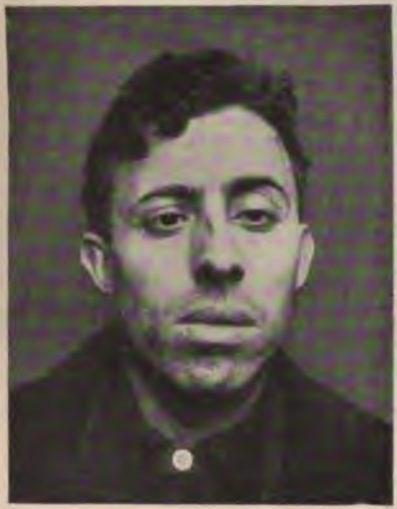
By G. L. WALTON, M.D.

The terms degeneration and degeneracy in their academic sense denote a lowering in the scale of being, particularly with regard to moral qualities. The custom has crept in to apply them to all evidences, physical or mental, of deviation from the average normal, and to call the possessor of such evidences degenerate. The term superior degenerate, originally introduced to designate the individual with mental deviation, has been extended to include the possessor of minor signs of deviation, whether physical or mental. This use of language is unfortunate, since it implies a conclusion by no means established, and conveys an opprobrium, lessened, it is true, by increasing familiarity, but in the majority of instances quite inappropriate. The desirability of introducing the purely descriptive terms, deviation and deviate, will be considered later.

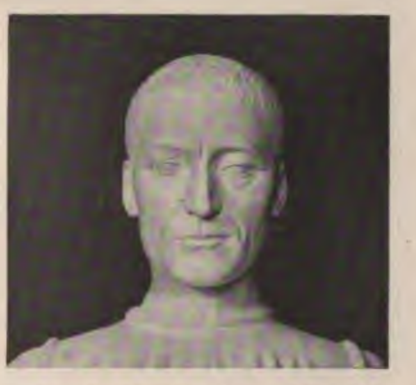
This subject, aside from its general importance, is of special interest to the neurologist, since the so-called stigmata of degeneration have to be constantly taken into consideration as bearing on the diagnosis and prognosis of nervous disorders.

To the oculist the subject is of equal interest for two reasons. In the first place, the so-called stigmata of degeneration, a long list of which will be found at the end of the chapter, include a large number of ocular defects, such as astigmatism and other errors of refraction, difference in color of irides, flecks on the iris, coloboma, and albinism, besides such functional disturbances of vision as night blindness and achromatopsia. In the second place, it is of practical importance for the oculist, as well as for the neurologist, to recognize these signs, since they often indicate the constitution and character of the individual, whatever relation they may bear to degeneration. For in estimating the relation of eye-strain to headache or other nervous disturbances, it must be remembered that such symptoms may be constitutional as well as reflex.

Among the most obvious and most uniformly listed "stigmata of degeneration," facial asymmetry comes first. A common form, but



1.—Common form of facial asymmetry, but unusual in degree.



po Strozzi by Baudetto da Maiano. (Berlin Museum.) Right side of face appears longer.

[illegible] patently [illegible] A [illegible] percentage of [illegible] has [illegible] A [illegible] difference in [illegible] in length of [illegible]

[illegible] of the face, even [illegible]

[illegible] is not easily explained [illegible] of material [illegible] fairly well [illegible] knowledge of the [illegible] from the others. [illegible] be justified in [illegible] has been reckoned [illegible] in a circle to deem [illegible] any of the peculiarities [illegible] signs of degeneracy.

[illegible] but equally insignificant. [illegible] This peculiarity has [illegible] be named the [illegible]

[illegible] of the lower part [illegible] external auditory meatus [illegible] with the alveolar process [illegible] from the root of the [illegible] angle between these

---

[illegible] eye is generally [illegible] photographed behind [illegible] characteristic of left- [illegible] writer that he [illegible] due to mechanical [illegible] the many hundred [illegible] with reference [illegible] the writer, facial asymmetry [illegible] average individuals.

[illegible]

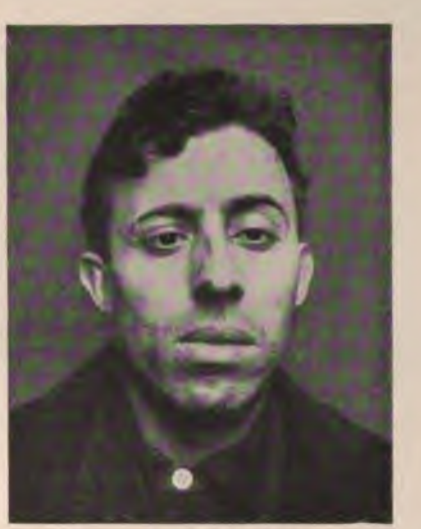


FIG. 1.—Common form of facial asymmetry, but unusual in degree.

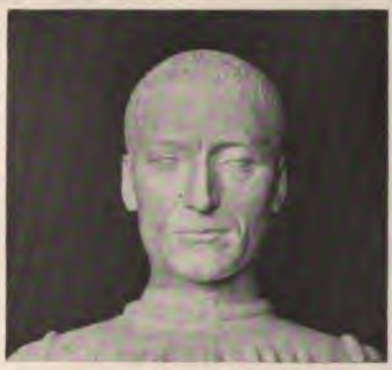


FIG. 2.—Bust of Filippo Strozzi by Baudetto da Maiano. (Berlin Museum.) Right side of face appears longer.

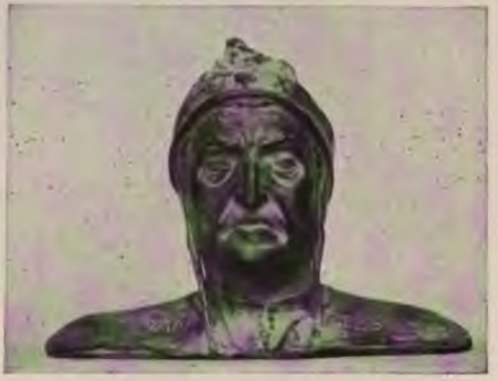


FIG. 3.—Bust of Dante. (From bronze in Naples Museum.) Left face appears longer.

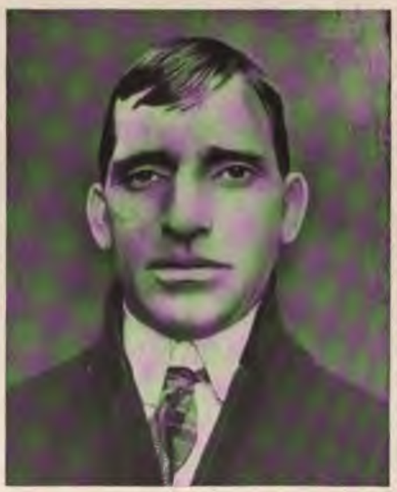


FIG. 4.—Less common form of facial asymmetry.

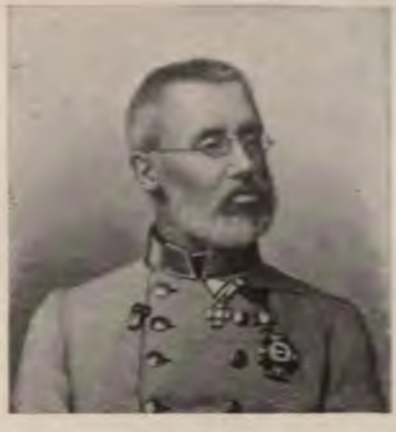


FIG. 5.—The "Hapsburg lip," as shown by Albert, Archduke of Austria. An example of deviation, not degenerate.

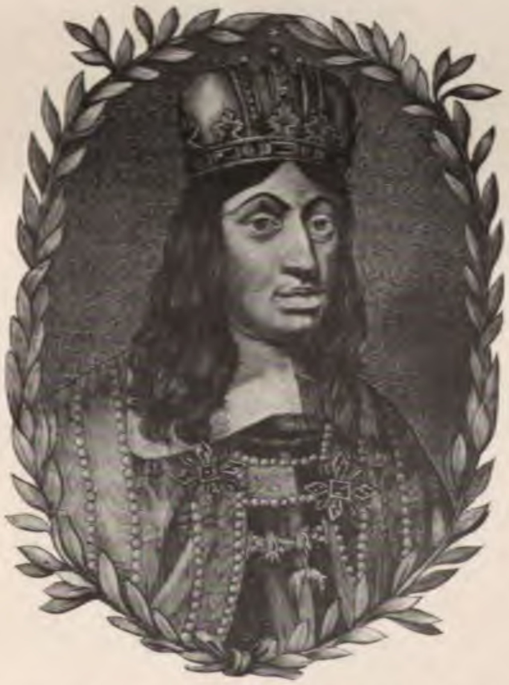


FIG. 6.—The same family peculiarity as shown by Leopold I.

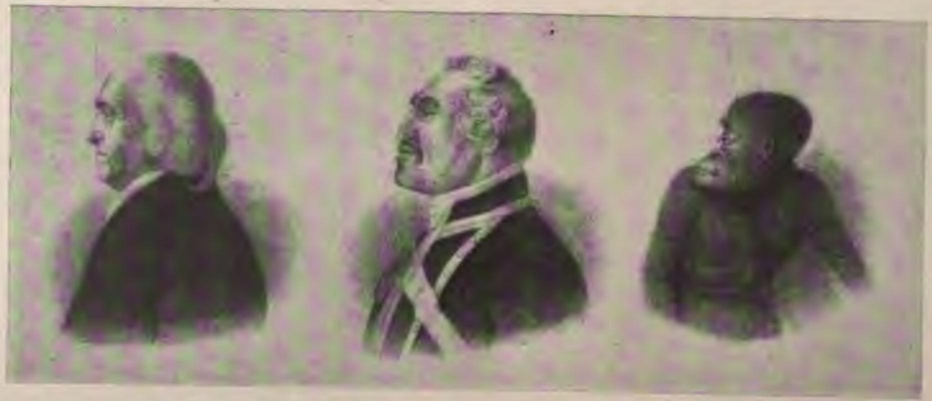


FIG. 7.—From an old print (published May 1, 1832, by John Brooks, 421 Oxford Street, London) illustrating the prognathism of Ferdinand VII., whose facial angle falls nearly midway between that of Jeremy Bentham and that of the orang-outang.

lines, or by the angle between the latter line and a vertical dropped from the root of the nose to the horizontal.

The accompanying old print (Fig. 7) shows the deviation of the facial angle of Ferdinand VII. from the ideal, as represented by the profile of Bentham. It seems probable that this is classed more appropriately under the stigmata of degeneration than simple facial asymmetry, though even this point is not really established. It was stated in the original legend that this represented the prognathism generally found in kings!

The illustration of Charles V. of Spain (Fig. 8) presents a further example of the Bourbon prognathism.

In the case of Charles V. there was also projection of the lower teeth, and it is said this malformation of the jaw seriously impeded mastication.

Ears badly set, asymmetrical, of unusual shape, lacking normal markings and normal parts, fall into the category of deviations which have been grouped as degenerative, but like most of the so-called stigmata, each peculiarity deserves separate study before accepting this sweeping dictum. For example, ears set too low, or lacking lobes and markings, may, perhaps, be degenerative, while a prominent antihelix or a moderate difference in length may have no such significance.

It is not, however, the purpose of this paper, to group this long list of deviations with regard to their bearing, or lack of bearing, on the question of degeneration. The illustrations cited will suffice to show that the estimation of their varying significance requires detailed and exhaustive study. This study is of practical importance in that it imparts knowledge regarding the constitution of the individual, a knowledge which helps the physician to weigh symptoms. Whichever of these deviations, alone or in combination, represent degeneration, it is true that they are generally more marked and more abundant in the insane, the criminal, and the idiotic, than in the well-balanced and the moral. This is, in fact, the important argument for attributing to all deviations the stigma which belongs only to a part, and to that part in an uncertain degree. It is also true that signs of deviation are common among those possessing signs of inherited nervous and mental instability as well as hysteria, epilepsy, hypochondria, and other psychical and physiological evidences of constitutional peculiarity or defect. In fact, these tendencies have been included in the list as psychical and physiological stigmata of degeneration.

Given, therefore, a case in which any of these symptoms are suspected of resulting from eye-strain, it behooves the practitioner to be on the lookout for other signs, both physical and mental, of deviation, for the more he finds, the greater is the probability that the symptom in question is due, in part or in whole, to the constitutional makeup of the individual, rather than simply to reflex irritation.

THE BEARING OF CONSTITUTIONAL DEVIATION UPON THE ESTIMATION OF EYE-STRAIN SYMPTOMS.

The study of the relation between eye-strain and symptoms referable to the nervous system has been handicapped by the apathy, not to say incredulity, on the part of the patient, general practitioner, neurologist, and sometimes even oculist, regarding the variety and extent of symptoms which may be produced by the continuous, though apparently insignificant, irritation resulting from moderate refractive error. (See Chapter XVIII.)

On the other hand, before predicting relief of nervous symptoms by correction of refraction, one definite factor in diagnosis must be carefully estimated, namely, the constitution of the individual. *In a considerable number of cases the error of refraction is merely one evidence of a faulty organism, among other signs of which appear headache, the invalid habit, vertigo, indigestion, irritability, and allied nervous states, to say nothing of choreic movements, epilepsy, and other motor phenomena whose relation to eye-strain is not yet definitely determined.* While many of these symptoms are sometimes solely dependent upon eye-strain, hence susceptible of amelioration by glasses, and while the adjustment of glasses is often followed by gratifying results even in the constitutionally defective, a certain proportion of cases will baffle the most skilful and painstaking ocular treatment.

With a view of emphasizing the practical bearing of this subject, it seems appropriate at this point to refer to a few of the most important and constant symptoms requiring analysis by the neurologist and the oculist, with reference to eye-strain as an etiological factor.

The most constant and important of these conditions is *cephalalgia*, a symptom frequently relieved, and even cured, by simple correction of refractive error. To Dr. S. Weir Mitchell is due the credit of first calling general attention to this relation, a relation apparently already recognized in practice by various oculists.

It is, however, not unusual that a sufferer from persistent headache is forced to apply, in turn, to the oculist, the rhinologist, and the gynæcologist. Examination discloses, successively, an error of refraction, a deviated septum, or projecting turbinate, and a misplaced or otherwise abnormal uterus; but each successive special treatment, though encouraged by definite pathological findings, results in only temporary and insignificant relief of the headache. In such a case *the underlying constitutional defect has been overlooked.* This headache is often vaguely localized, often vertical, is less apt to be limited to the forehead and the occiput, and is less often distinctly migrainous (see Chapter XVII.) in seat and character than that of the purely reflex ocular variety.

The exact nature of the constitutional headache cannot be determined. It seems improbable that there is a definite physical basis. In such event we are forced to resort to the unsatisfactory expression "functional." In some cases the description of suffering is so much at variance with the appearance and demeanor of the patient that the examiner is forced to the conclusion that the so-called suffering is an interpretation put by the patient upon what, in another, if noticed at all, would be merely a discomfort; in other words, that the normal pressure of part upon part of the cranial contents, or the normal changes in circulation of the various fluids, or the sensation accompanying normal mental effort, phenomena unnoted by the average individual, may be acutely and uncomfortably felt by the highly sensitive possessor of the psychopathic temperament.

A well-recognized analogy is found in tinnitus aurium, a symptom generally due to aural disease, and often susceptible of relief by aural treatment, but resulting sometimes (in the morbidly sensitive or hysterical individual), from the mere irritability of the sensorium, unable to accustom itself to the physiological impressions received by the auditory nerve.

This variety of headache has been described and classified by writers on degeneracy as *constitutional headache*, and must be recognized as a sign of defective nervous makeup, of which the refractive error and the nasal and pelvic anomalies furnish merely contributory evidence.

Another symptom not infrequently relieved by glasses is *nervous irritability.* Here again the practitioner should strive to distinguish between simple nervousness produced by continuous peripheral irritation, and the various mental peculiarities resulting from inherent, and inherited, tendencies. The practitioner is often made aware of the mental attitude of patients of the latter class by a peculiar fussiness, by complaints of the temperature of the room, by a call for a fan, a glass of water, the shading of a light, the closing of a window. They often adopt an antagonistic attitude, and question the need of the various steps in the examination. Such patients, while complaining of most excruciating torture, are often unwilling to make the sacrifice of wearing glasses for its relief, or if they make the trial are unable to accustom themselves to the bows over the ears, the sight of the rims, or the reflection from the glasses. The efforts of the oculist are still further handicapped by the inability of such patients to relax the muscles of accommodation after the adjustment of glasses. They are also peculiarly prone to attempt extension of vision beyond its legitimate limits, and to look from side to side, though the latter tendency may be, in a measure, somewhat counteracted by terescopic or toric lenses.

While the psychopath describes his headache in most extravagant terms, no corresponding signs of physical suffering appear. Though complaining of utter collapse, such a patient will often complete a long examination with smiling countenance and showing less sign of exhaustion than the examiner. The history in such cases will often disclose also hypochondriacal tendencies, morbid fears, antipathies, hyperconscientiousness, or mysophobia (fear of contamination). A combination of mental traits pointing in the same direction has been well summarized by Soukhanoff[3] under the "Ideo-Obsessive Constitution."

Individuals with this peculiarity are easily recognized. They are "over-scrupulous, disquieted over trifles, indecisive in action, and anxious about their affairs. They are given early to morbid introspection, and are easily worried about their own indispositions or the illnesses of their friends. They are impressionable, possess considerable *amour propre*, and are easily offended; but as they are usually reticent they hide their resentment and are often secretly ashamed of those bizarreries peculiar to their disposition. They are often timorous and apprehensive, and prone to pedantism. The moral sentiments are pronounced in most cases, and if they are, as a rule, somewhat exigent and egotistic, they have a lively sense of their own defects."

Hypochondria has been made a special subject in another chapter (XVI.), but deserves further brief mention in this connection. Hypochondria implies undue attention to symptoms, and undue alarm concerning them; it may exist alone or in combination with other diseases, organic or functional. This tendency shows itself, as a rule, early in the investigation. The visit of the hypochondriac is apt to be preceded by a complete history of his case in writing. He is likely to consult memoranda in the elaboration of this history, and to describe his symptoms with great particularity. He is prone to describe his sufferings in anatomical terms; for example, instead of stating he has a pain in the side, he locates the pain in the lung, the heart, or the intercostal nerve. He comes to the examiner with a preconceived and unshakable diagnosis of his own case, and in its defence exhibits great argumentative ingenuity. He is generally anxious and willing to try a new remedy, usually with the result of temporary improvement. After the patient's departure the physician may expect to receive letters containing further elaboration of symptoms.

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[3] Rev. Neurolog., Juin 30, 1903, p. 613. Abstract in Review of Neurology and Psychiatry, October, 1903, p. 703.

These psychoneuroses, still often loosely included under the title of neurasthenia,[4] are not to be mistaken for results of eye-strain, though marked refractive error is extremely likely to appear; and while no effort should be spared to contribute to the comfort of the patient by removing every possible source of reflex irritation, the chance is remote of overcoming inherent tendencies.

Another condition for the relief of which glasses are not infrequently recommended is *epilepsy*. The repeated failure of this procedure is undoubtedly due to the fact that in the vast majority of cases this disease represents a constitutional defect in the cerebral mechanism. (Compare Chapter XVIII.)

If experience had shown that overuse of other muscles than those of the eye produced generalized convulsions with impairment of consciousness, the burden of proof would be upon those denying that epilepsy is a common result of eye-strain, but in the absence of such evidence overwhelming proof is required to fortify the position of those who support this proposition.

In the overuse of other muscles (for example, those of the forearm and hand concerned in writing), if spasm appears it is practically limited to the muscles involved; there is certainly no generalized spasm; nor is there a suggestion of impaired consciousness, the one essential symptom of epilepsy. In eye-strain the analogue of the local cramp is found in the spasm of accommodation, a well-recognized symptom. It is true that the analogy between eye-strain and writers' cramp is not complete in every particular, inasmuch as the pain of writers' cramp, in contradistinction to that of eye-strain, is usually limited to the overused member, and generally appears during, or immediately after, the overuse of the part. The divergence from this rule in the case of pain resulting from eye-strain is, however, insignificant compared with the introduction of such new elements as generalized convulsions and impairment of consciousness.

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[4] See Dana, The Partial Passing of Neurasthenia.

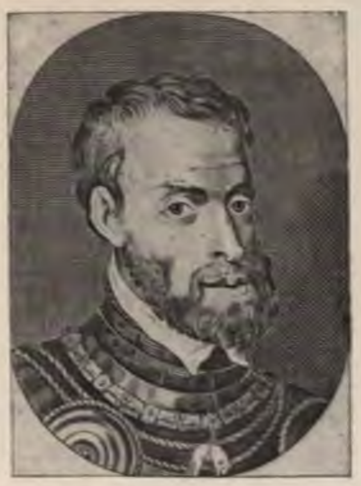

FIG. 8.—A further illustration of the prognathism of the Bourbons, as shown by Charles V. of Spain.

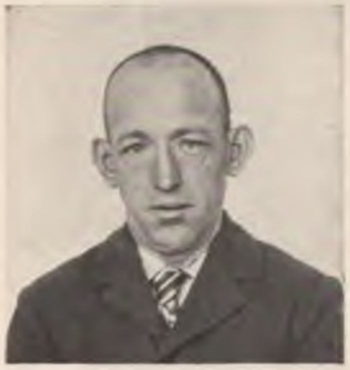

FIG. 9.—Ears asymmetrical, badly shaped, badly set. (From Massachusetts School for Feeble Minded, through kindness of Dr. Fernald.)

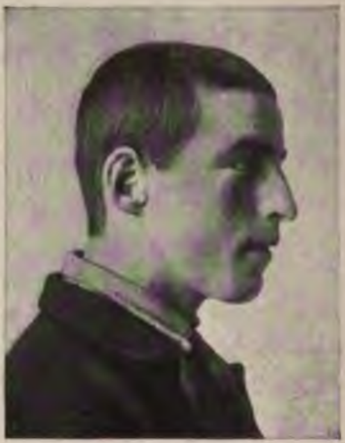

FIG. 10.—Absent lobe, crumpled helix, antihelix prominent and of unusual shape. Incisoura intertragica broadened, concha large. (Same source as Fig. 9.)

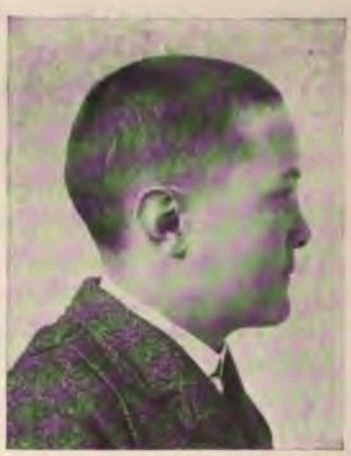

FIG. 11.—Helix narrow, thin, and irregularly shaped, with suggestion of Darwinian tubercle; antihelix prominent; abnormal fold in lobe. (Same source as Fig. 9.)

blinking, or sniffing, mannerisms in speech and deportment are suggestive. Among the *physical* peculiarities may be noted deviations in form of the skull and face,[5] unusual shape, size, or position, of the orbits, narrow palpebral fissure, excessive prominence of the forehead, prognathism and projection of the lower teeth (Figs. 7 and 8), peculiarity in shape of the palate, peg-shaped incisors, hair upon the face (in the female), ears badly shaped, unlike, or badly set (Figs. 9, 10, and 11), and curvature of the spine. It must not be understood that the physical and mental stigmata always go hand in hand.

DEVIATION.

The various constitutional defects and peculiarities have been summed up, as has been said, rather loosely, under the term degeneracy or degeneration. These stigmata include every conceivable variation from the average normal (Dana, Church and Peterson, and others), ranging all the way from such innocuous peculiarity as a difference in color of the two eyes, to perversion and imbecility. It has not, however, been yet established that every sign of deviation from the average normal is degenerative, and it seems to the writer [6] desirable to name the phenomena signs of deviation and to call their possessor deviate, or a deviate, as the case may be, limiting the term degeneration only to such deviations as obviously imply deterioration, either of the individual or of the race. The nomenclature which has hitherto obtained in anthropological study, largely through the writings of Nordau [7] and of Lombroso,[8] if applied to the vegetable kingdom, would without investigation stigmatize the four-leaved clover as degenerate.

The illogical nature of the present method is illustrated by the statement of Lombroso [9] that four per cent. of *normal individuals* have five or more *signs of degeneration.* Would it not be equally descriptive and more accurate to say that this percentage of normal individuals have signs of deviation?

BIOGRAPHICAL STUDY OF CONSTITUTIONAL DEVIATIONS.

A careful study of the lives of various men prominent in literary and scientific work would indicate that they were hampered by eye-strain. (Gould.[10]) But the same study made with due consideration for possible etiological factors other than eye-strain, for the mental and physical discomforts under which they labored, shows that the problem is not so simple. It is a question whether enthusiasts in other branches of medicine might not find ample evidence of the injurious effects of pathological states involving other organs. Might not, for example, the dentist attribute De Quincey's indigestion to his defective teeth? Or, again, might not the diathetic enthusiast find sufficient evidence in Carlyle's case to claim an excess of uric acid as the cause of his unfortunate disposition? However this may be, the history of such cases is apt to reveal signs, physical, mental, or both, of constitutional peculiarity. Carlyle in his boyhood was shy, proud, pugnacious, with strong affections and with equally violent antipathies. In later life he declared war upon the neighbor's poultry, and had constructed a room for his labors, proof against sound. De Quincey was a man of diminutive stature, given to day-dreaming before he became addicted to his unfortunate habit, the inability to resist which may quite as plausibly be attributed to constitutional defect as to the discomfort caused by eye-strain.

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[5] The usual form of asymmetry, previously discussed, has probably no bearing.

[6] Walton, The Prevailing Conception of Degeneracy and Degenerate, with a Plea for Introducing the Supplementary Terms Deviation and Deviate. Boston Medical and Surgical Journal, January 21, 1904.

[7] Degeneration, Max Simon Nordau. New York: Appleton & Co., 1895.

[8] L'uomo delinquente ecc., 1889.

[9] Lombroso, in Twentieth Century Practice of Medicine. New York, 1897, p. 381.

The exalted character of the many celebrities cited by Lombroso,[11] in establishing his conclusion that genius is a degenerative psychosis, accentuates the necessity of supplementing the word degeneracy by some less opprobrious, while equally descriptive and inclusive term. Even the fact that many of these individuals showed decided deterioration in the physical or moral scale does not militate against adopting a term for the classification of their variations from the normal before drawing conclusions regarding the significance of those variations. In dealing with genius it is well to remember also that the impression prevailing in popular, not to say scientific, circles, that moral qualities stand in an inverse ratio to intellectual endowment, has been controverted by the statistical investigations of Woods.[12]

Etiology.—The varied *etiology of the so-called stigmata of degeneration* would already indicate great diversity in their downward, negative, and even occasionally upward values. The wide and indiscriminate use of the word degeneration has led to conflicting views of the origin of the stigmata. Critical review of this etiology is as far beyond the ability of the writer as beyond the scope of this treatise, but brief mention of some of the suggested causes will be in place. From Nordau's point of view it is natural that disease, privation and excesses in the ancestry should be regarded the main factors in the production of degeneracy, and it is doubtless true that such causes result in enfeeblement, undersize, and defective resistance in the progeny; but according to modern scientific views the presence or absence of inherent characteristics transmitted by the germ are independent of such influences. In other words, acquired characteristics are not transmitted.

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[10] Biographic Clinics. Philadelphia: P. Blakiston's Sons & Co., 1903.

[11] The Man of Genius. London: Walter Scott, 1901.

[12] F. A. Woods, The Correlation between Mental and Moral Qualities. Popular Science Monthly, October, 1903.

The present anomalous use of the term degeneration includes among the deviations from the average normal, even such evolutional spurts as unusual development of Broca's convolution in the individual possessed of unusual linguistic aptitude (Lombroso). This use of the term renders inadequate the etiology of Nordau.[13] Paul Jacoby[14] has endeavored to show that selective tendency in aristocratic circles is answerable for sterility and family decadence. Similarly, Ireland[15] has laid stress on indolence and the "insanity of power" as causing degeneracy in royal families. The studies of Woods[16] clash with these conclusions, for he has shown by the analysis of many royal families, in which the surroundings and manner of life remain practically constant factors, that while some families have deteriorated others have advanced, the variations in the individuals and families depending preeminently on the combinations of characteristics inherited.

Classification of Causes of Deviation.—I. Modern embryological investigations have shown that *the inherent and therefore hereditary characteristics transmitted through the germs entirely dominate the structure of the progeny.*[17] Just what characteristics the child will inherit from each parent cannot be foretold; still less can it be predicated what combinations of more remote ancestral traits will appear in any given progeny. For the study of this fundamental branch of the subject the views receive general acceptance or not, this line of thought will serve to remind us that the absence of neurotic history in the immediate family does not preclude such inheritance from a distant ancestor. It is possible that Mendel's laws of dominance and recession (latency) may prove of aid in determining the reappearance of deviative signs.

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[13] Prior to Nordau's more popular treatise, Morel (Traité, etc., 1857, p. 17) had dwelt upon the constitutional basis for insanity.

[14] Etudes sur la Selection dans ses Rapports avec l'Hérédité chez l'Homme, Paris, 1884.

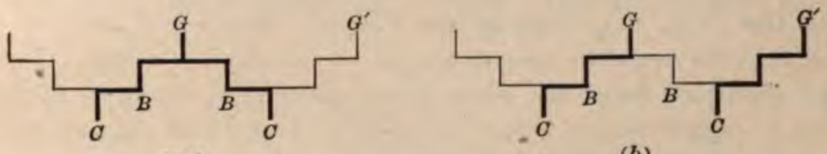
[15] The Blot upon the Brain. G. P. Putnam's Sons, New York, 1896.

[16] Mental and Moral Heredity in Royalty. Popular Science Monthly, August, 1902, to April, 1903.

[17] Wilson summarizes the present view of maturation of the sexual elements thus: "In both sexes, the final reduction in the number of chromosomes is effected in the course of the last two cell divisions by which the definite germ cells arise, each of the germ cells thus formed having but half the usual number of chromosomes. In the female but one of the germ cells forms the ovum proper, while the other three, known as the *polar bodies*, are minute, rudimentary and incapable of development." "In the male, on the other hand, all four of the cells become

(*b*) The view long accepted without question that the effect of consanguineous marriage in itself is harmful is voiced by Mercier, who states that while a certain degree of dissimilarity between parents produces the best offspring, *too great similarity in parents* tends to enfeeblement and idiocy, while too great dissimilarity, if productive at all, tends towards wildness in the offspring. It is, he says, on account of the deleterious effect of parental similarity that marriage between near relatives is undesirable, but if it should chance that cousins obtain their prepotent qualities from different ancestors rather than from the same ancestor, the liability to deterioration in the offspring is lessened.

If the prepotent qualities are represented by heavy lines in Fig. 12, the cousins *C*, *C*, children of brothers *B*, *B*, inherited, in (*a*), prepotently the characteristics of this common (paternal) grandfather, *G*, hence their marriage, according to Mercier, would be unwise. One of the cousins in (*b*), however, derives prepotent qualities from the maternal grandparent (G^1), so that the risk to posterity is reduced to the minimum.



(*a*) (*b*)

FIG. 12.—After Mercier.

This view of the deleterious effects of close intermarriage is not borne out by the extended investigations of A. H. Huth,[25] excepting in so far as the cousins inherit in common *vicious tendencies.*

Whatever evil effects may follow continued inbreeding, they may be materially reduced by the introduction of new blood after several generations, as in the case of the most notable instance on record of continued consanguineous parentage, namely, that of the Ptolemies. (Mercier.) The repeated recurrence of the Hapsburg lip (Figs. 5

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[24] Castle, Proceedings Amer. Acad. of Arts and Sciences, vol. xxxviii. No. 18. With bibliography. Cambridge, Mass., January, 1903.

[25] The Marriage of Near Kin, etc. London, 1887.

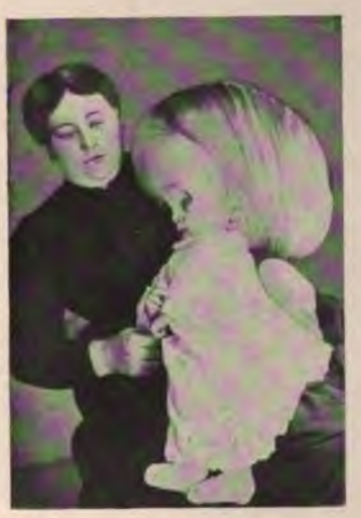

FIG. 13.—A remarkable degree of hydrocephalus. (Copyright, 1888, by Langill, Hanover, N. H.)

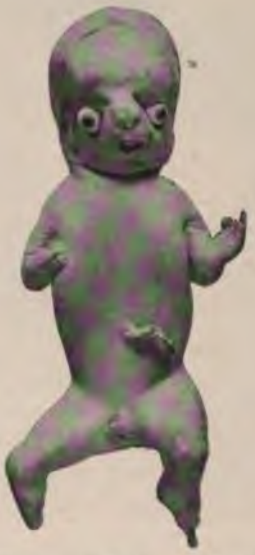

FIG. 14.—Amputation by strangulation of fœtal cord. (From cast in Warren Museum.)

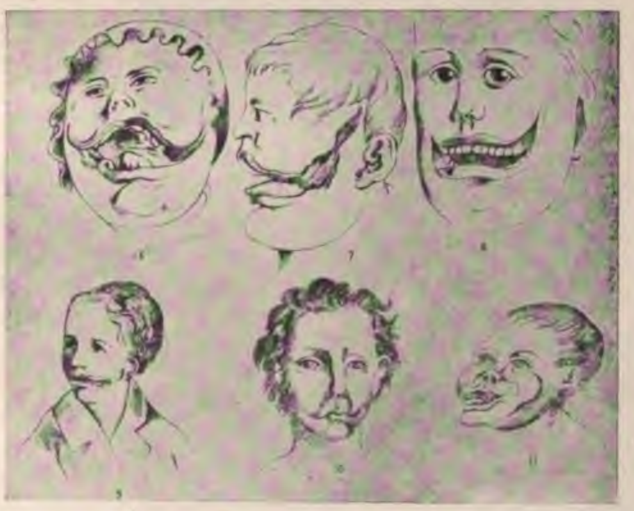

FIG. 15.—Results of amniotic adhesions. (From plates in Ahlfeld, Misbildung des Menschen 1880.)

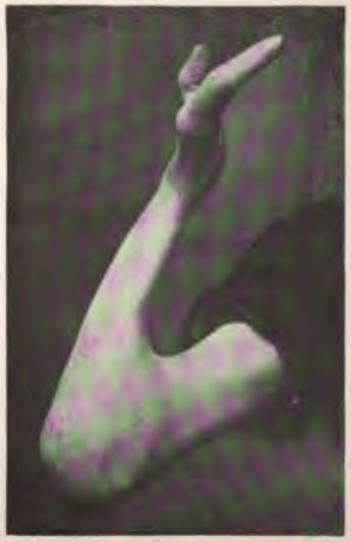

FIG. 16.—Mandible hand; absence of three outer digits and of ulna, with webbing at elbow; isolated defect.

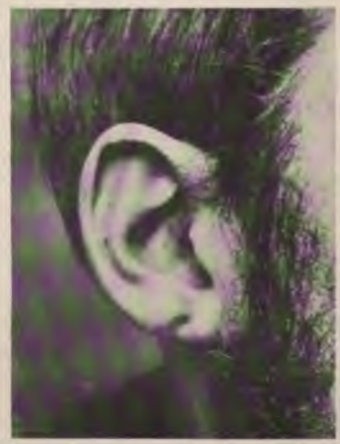

FIG. 17.—Defect in ear recurring in family.

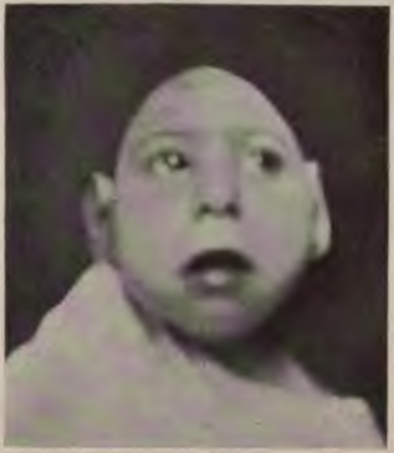

FIG. 18.—Shape of skull resembling that produced by moderate inflation in Thompson's experiment.

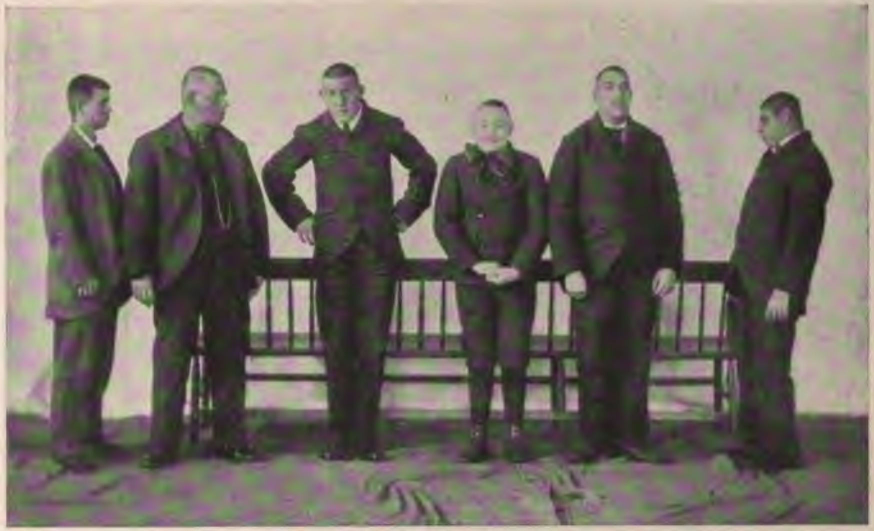

FIG. 19.—Group of microcephalic inmates of Massachusetts School for Feeble Minded. (By kindness of Dr. Fernald.)

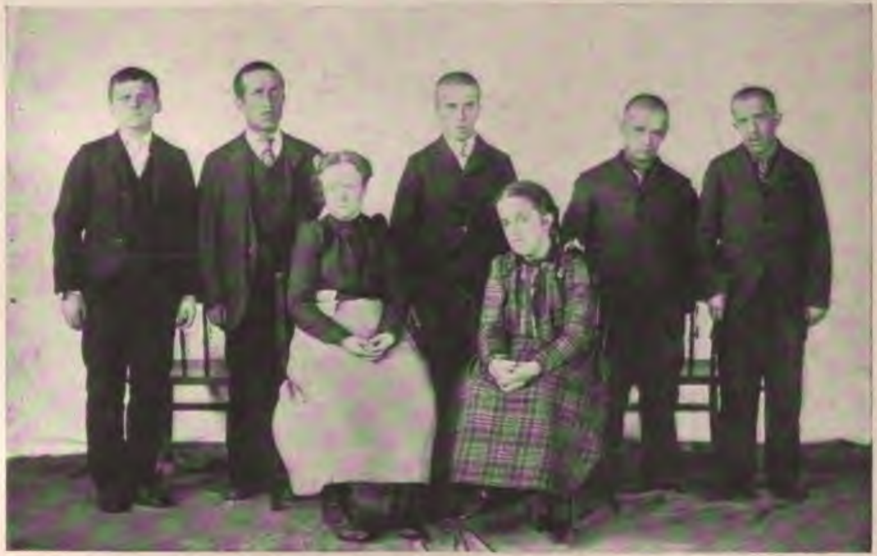

FIG. 20.—Group of macrocephalic inmates of the same institution.

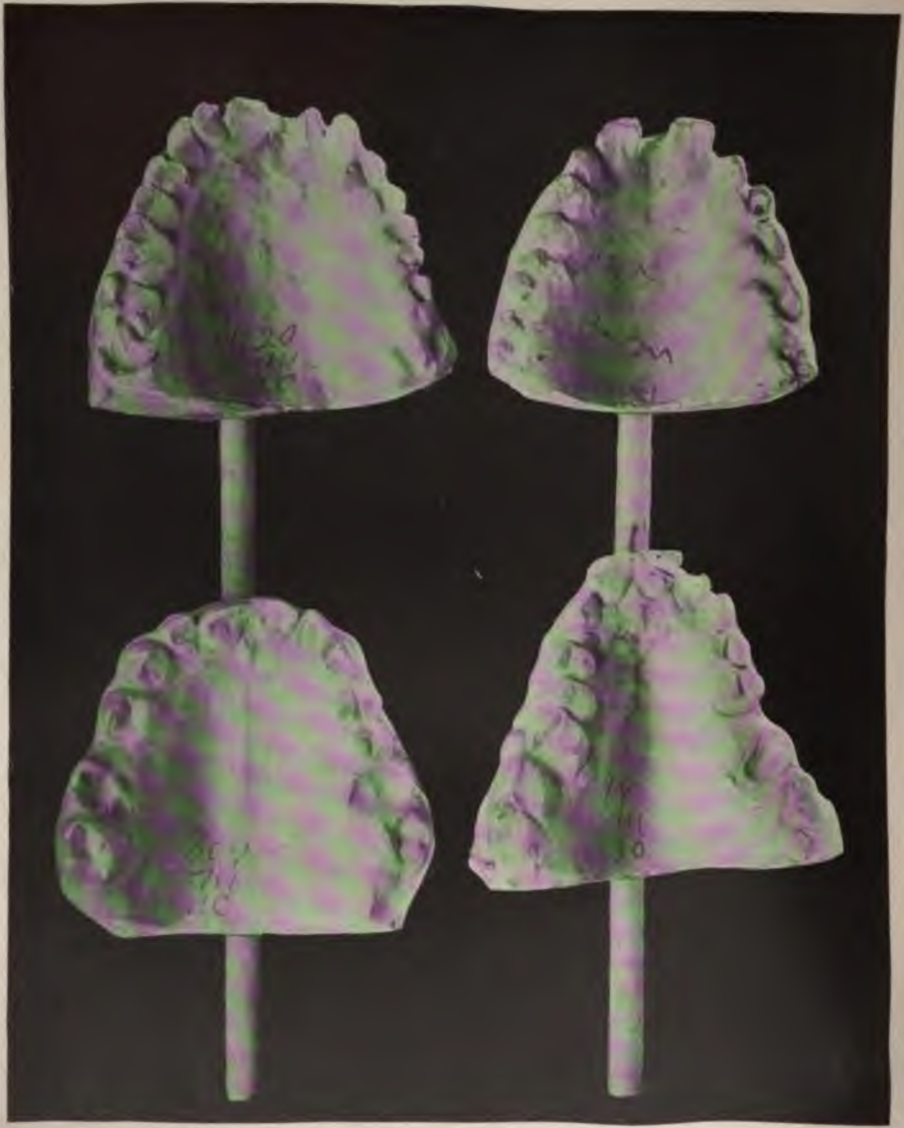

FIG. 21.—Casts of palates from the collection of Dr. Channing. They illustrate varieties in shape, width, height, and symmetry; one shows "torus palatimus;" none are absolutely normal. All are taken from the feeble minded.

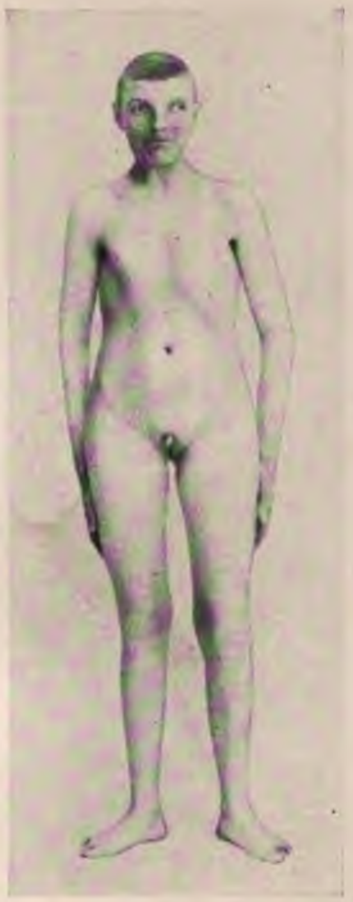

FIG. 22.—Inmate of the same institution, aged 23. Absence of hair on face and pubes. hypospadias, infantile penis, cryptorchismus, muscular insufficiency.

V. The investigations of Thompson tend to show that *mechanical influences affecting the development of the infant* should be given a place. He maintains that the shape of the skull depends largely on the cranial capacity, and in some degree also upon the strength of the temporal muscle. This investigator replaced the calvarium by a rubber bag to represent the membranous condition of this part of the skull during its developmental period; moderate inflation of the bag produced a shape resembling that of the ape's skull with slight frontal development, and, viewed from in front, a shape suggested by that of the idiotic child shown in Fig. 18 (for which I am indebted to Dr. Waterman).

Great distention produced the normal adult shape. This difference was accentuated by weight-bearing cords attached to the bag in the region of the temporal muscle. The question is complicated by the difference in leverage under which the temporal muscle works with different shaped jaws, the long arm of the lever being the condylosymphyseal measurement, the short, the condylocoronoid measurement. The similarity in shape of microcephalic and macrocephalic skulls respectively in the groups of feeble minded (Figs. 19 and 20) seems to lend weight to Thompson's hypothesis.

VI. It has already been stated that constitutional disease, deprivation, and excesses in the ancestry may so far impair the nutrition of the germ as to cause undersize, enfeeblement, and lessened resistance in the offspring though not influencing its inherent characteristics.

VII. Certain glands have a distinct, though not thoroughly understood, influence on the nutrition of the body; hypertrophy or defect of these glands produce marked variation in its growth. Giantism is found associated with enlarged pituitary gland, and results perhaps from its oversecretion; possibly dwarfism implies diminution of this secretion, though this point has not been investigated as far as the writer is aware. Acromegaly, a condition in which the face, jaws, hands, and feet become enormous in later life, after the bones have acquired their length, is also found associated with disease of this gland and deserves a place in the same category with giantism. Defective glandular tissue in the thyroid produces cretinism in infancy, myxœdema in adult life.

VIII. Dr. Minot (to whom I am indebted for critical revision of the portions of this chapter which bear on embryology) suggests that a fundamental etiological factor should be added, namely, such lack of development as is evidenced by harelip. He attributes the erroneous use of the word degeneration mainly to failure to recognize the essential steps in degenerative processes, embraced under *cytomorphosis.* This word he uses "to designate comprehensively all the structural modifications which cells, or successive generations of cells, may undergo, from the earliest undifferentiated stage to their final destruction."[28] In other words, existence of protoplasm and its growth are prerequisites for degeneration. A deposit of horny material in the skin may then be correctly classed as degenerative, in contrast to such conditions as harelip. Nor can degeneration of the race be established upon signs which do not indicate degeneration in the individual.

It would seem, then, that we must include among the possible causes of the so-called stigmata of degeneration (which include signs of deviation only), at least

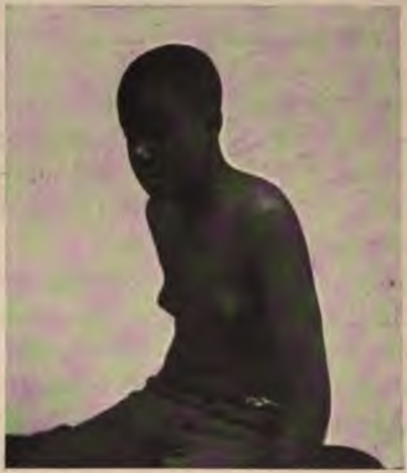

FIG. 23.—Unusual development of the breasts in a negro boy. (Case of Dr. Spiller.)

I. The potential variations from the average normal contained in the parent germ, including the results (*a*) of atavism, (*b*) of parental similarity, and (*c*) of selective tendency on the part of the ancestry.

II. Intra-uterine infection.

III. Mechanical injury during intra-uterine life.

IV. The absence or peculiarity in the germ of certain elements, or their disappearance or anomalous development, without traceable inherited tendency or other known explanation. To this class of stigmata the unsatisfactory and doubtless inadequate term "freak of nature" (lusus naturæ) has been applied.

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[28] A Laboratory Text-Book of Embryology. Philadelphia: P. Blakiston's Son & Co., 1903.

V. Mechanical influence exerted during infancy.

VI. Deleterious influences and habits in the ancestry (productive of enfeeblement, undersize, and lessened resistance in the progeny but not altering the essential potential characteristics transmitted by the parent germ).

VII. Absence or hypertrophy of certain glands (pituitary, thyroid) which have a nutritional influence.

VIII. Arrest of development such as is seen in harelip and similar defects.

List of Stigmata.—The following list comprises a large number, though by no means all, of the pathological conditions and anomalies which have been cited by various authorities as stigmata of degeneration. It is obviously more appropriate to classify a considerable number of them as signs merely of deviation. Some of those whose presence, either alone or in combination with others, have little if any bearing on the question of true degeneracy are starred.

ANATOMICAL STIGMATA.

Cranial Anomalies.

- Asymmetry of skull, especially frontal bone.
- Macrocephalus.
- Microcephalus.
- Platycephalus or chemocephalus (flat).
- Leptocephalus (narrow).
- Oxycephalus or acrocephalus (steeple shaped).
- Plagiocephalus (oblique).
- Scaphocephalus (keel shaped).
- Trigonocephalus or trigonal (pointed anteriorly).
- Short parietal arc.
- Short frontal arc.
- High prominent forehead in women.
- Heavy jaws.
- Great or unequal prominence of malar bones.
- Lemurian hypophysis.
- Prognathism.
- Orthognathism.
- Opisthognathism or retrognathism.

  } Unusual projection, straightness or recession of the lower face.
- Large frontal sinuses, small orbit.
- Crania progenæa (lower teeth projecting beyond upper, and inferior maxillary angle obtuse).
- *Facial asymmetry.
- Anomalies of the eye.
  - Narrow palpebral fissure.
  - Micropthalmus.
  - *Flecks on the iris.
  - *Chromatic asymmetry of the iris.
  - Albinism.
  - Congenital cataracts.

ANOMALIES OF THE EAR (CONTINUED).
- Asymmetry of the two ears, general anomaly of left (Blainville ear).
- *Prominence of antihelix.
- *Adherent lobules (found in a large percentage of normal individuals).

[Definite types of misshapen ears are described and named, for example, the Stahl, Nos. 1, 2, and 3; the Wildermuth; the Morel; the Cagot. Descriptions and illustrations of these varieties may be found in the text-book of Church & Peterson, p. 628 *et seq.*]

Anomalies of the Limbs.
- *Left arm and leg longer than right.
- *Excessive length of arms.
- *Long fingers.
- *Polydactyly.
- Symphysodactyly or achistodactylus (joining of fingers).
- Syndactyly (web fingers).
- Ectrodactyly (fingers wanting).
- Amelus or ecromelus (limb wanting).
- Phocomelus (segment of limb wanting).
- *Megalomelus.
- *Megalodactyly.
- Oligomelus (excessive gracility).
- Oligodactyly.

Anomalies of the Trunk and General Structure.
- Hernias.
- Malformation of the breasts and thorax.
- Dwarfism.
- Giantism.
- Infantilism.
- Femininism in men.
- Masculinism in women.
- *Spina bifida.
- Feebleness of construction.
- Lordosis.
- Scoliosis.
- Kyphosis (Fere).
- Malformed coccyx.
- Thoracic asymmetry.
- Mammary development in males.
- Mammary absence or reduplication in females (polymastia).

Anomalies of the Genital Organs.
- Small or deformed genitals.
- Hermaphrodism.
- Cryptorchismus.
- Hypospadias.
- Epispadias.
- Defect, torsion or great volume of prepuce.
- Imperforate meatus.
- Labia too large or too small.
- Clitoris large.

Anomalies of Sensory Function (Continued).
  Nyctalopia (day blindness).
  Concentric limitation of the visual field.

Anomalies of Speech.
  Mutism.
  Defective speech.
  *Stammering.
  *Stuttering.

Anomalies of Genito-Urinary Function.
  Sexual irritability.
  Impotence.
  Sterility.
  Urinary incontinence.
  Amenorrhœa.

Anomalies of Instinct or Appetite.
  Mercyism (gluttony).
  Rumination.
  Uncontrollable appetites (food, liquor, drugs).

Diminished Resistance against External Influences and Diseases.
Retardation of Puberty.
Deficient Vital Activity of Organic Functions.
  Weak heart.
  Low arterial tension.
  Coldness of extremities.
  Flushing of extremities.
  General chills and *flushes.*
  Weak digestion.
  Constipation.

PSYCHIC AND PSYCHO-NEUROTIC STIGMATA.

  Dementia præcox.
  Compulsive insanity.
  Manic depressive.
  Melancholia.
  Hysteria.
  Hypochondria.
  Phobias.
  Psychopathic endowment.
  Invalid habit.
  Idiocy.
  Feeble-mindedness.
  *Eccentricity.
  Moral deliquency.
  Sexual perversion.
  Precocity.
  Overdevelopment of certain aptitudes.
  Paranoia.
  Ideo-obsessive constitution.

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